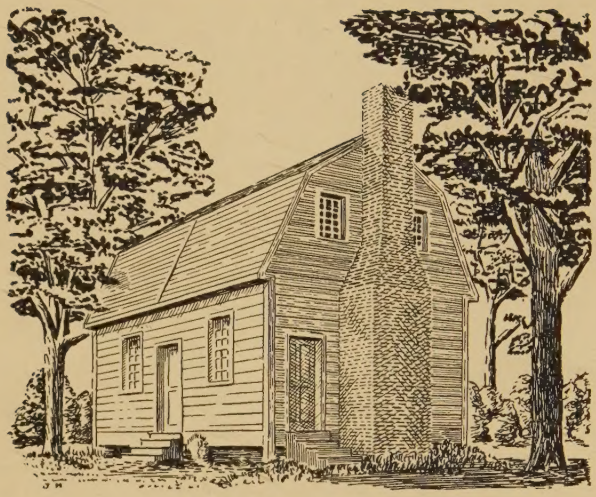

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DISEASES OF THE ARTERIES

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ANGINA PECTORIS

BY

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CHAPTER VIII

CARDIOSCLEROSIS AND MYOCARDIAL VALUES[1]

"Cardiosclerosis."—In all my papers, from the time that I began to write on these subjects, I have urged that in arteriosclerosis closer and more discriminating attention should be given to the conditions of the heart, and that our common phrases—for instance, that of "cardiosclerosis," or in still finer language "Myodegeneratio cardiosclerotica"—should be more considered. By the indiscriminate use of this term, the heart is brought into common condemnation with sclerosed vessels, a view which is only partially justifiable.

Huchard's Views.—We are in a position to consider the opinions of the late M. Huchard on this matter in their final form as, by request, he presented them finally in 1908 and 1909; in 1908 at the International Congress at Buda-Pest, in 1909 as "specially reported" by himself to a representative of the *Medical Press*.[2] On both occasions Huchard began by reiterating his postulate, that all arteriosclerosis, whether regarded as an anatomical lesion or as a clinical series, is one disease, a malady attacking heart, arteries, and kidneys as a whole; the affection of the heart being homologous with that of the arteries. In his view the heart goes down, not as vanquished in a strife against odds, but in a co-ordinated retrogression. The mere senile heart he explicitly withdrew from the category of arteriosclerosis. The whole malady of arteriosclerosis originates, so he declared, in certain intestinal toxins, pressor in action, which escape neutralisa-

[1] "Myocardial Values," the substance of an address to the Chelsea Clinical Society on March 12, 1912, under the title of "The Physician and the Pathologist on Heart Failure," published in the medical journals near that date.

[2] *Medical Press*, March 1909.

tion by the liver. The distinguished author went on to present a paradigm of the malady as a single and comprehensive morbid series, as follows :—

"First stage : 'Presclerosis, due to renal causes'—a 'cardio-renal' state with excessive pressures 'almost invariably.' The heart is affected by toxins from the very beginning ; the cardio-sclerosis, whatsoever its degree, dates from the initiation of the first stage, advances *pari passu* with the affection of the arteries, and is due directly to the intoxication."

"Second stage : Established myovalvular cardiosclerosis, together with the same depravation of the arteries, and both originating alike and together from the toxins."

"Third stage : The 'Mitro-arterial' (when mitral incompetency sets in)."

"Fourth stage : That of 'Cardio-ectasis,' a stage which may or may not be reached. Cerebral hæmorrhage, or other incident, may cut the life short. Cerebral hæmorrhage however, the alleged consequence of arteriosclerosis, occurs only in cases of chronic interstitial nephritis" (!).[1]

Auto-intoxication, cardiosclerosis, arteriosclerosis ! "Three words, Don Jorge ! and what may not be made out of three words ? You have lived among us to little purpose if you think we require more than three words to build a system with." But let us seek into the grounds of this fair-seeming edifice which seems at best to be speculative, and even at this to be inconsistent and equivocal. When it was urged that if many cases of arteriosclerosis are attended with high pressures, yet a large number persist from first to last with pressures little, if at all, above the ordinary, Huchard minimised the low-pressure cases as much as possible, retaining them in the same category but as transient phases, phases to be explained by cardiac reduction, or degrees of portal congestion with intermittence of that function of the liver which should neutralise intestinal toxins ; as in another place [2] he put it "toxins primarily of intestinal origin and associated with portal congestion." The

[1] These pages were written during Huchard's lifetime ; I desire now to qualify my criticisms by a tribute of great respect for the memory of a distinguished colleague, and of an earnest and devoted clinical physician.

[2] *Lancet*, Sept. 4, 1909, p. 720.

interviewer seems to have demurred, as regards the first or "presclerotic" stage, that there is often no evidence of renal or hepatic affection, perhaps much to the contrary; Huchard then took refuge in a vague summary of "renal causes mixed with abdominal toxins, and dietetic errors."

This is "Schematismus" indeed! The difficulties are (*a*) that in some 50 per cent of cases of arteriosclerosis exorbitant pressures are never manifested from first to last; many of them indeed being not inconsistent with fair or even good health up to advanced years; (*b*) that if we accept such a term as "cardiosclerosis," this should include the coronary and myocardial scleroses which occur no less in these low-pressure cases; (*c*) that in high-pressure cases—and these are they, be it remembered, which for Huchard made the main category of arteriosclerosis in the first stage of which the heart with its vessels should begin simultaneously to undergo degradation—the heart, instead of yielding, when pressures might fall, is so charged with intrinsic energy as to multiply its work, for a time out of its normal and intact reserves, and afterwards by the abundant hypertrophy which it still has at command; a development which may reach enormous dimensions, a robust capacity by which it succeeds for some years in concealing even from the patient all consciousness of the mischief on foot; (*d*) that only at length does this heart suffer defeat and begin to yield, with the usual consequences of this discomfiture, involving often of course the familiar forcing of the mitral valve; (*e*) that although in renal disease cerebral hæmorrhage is frequent, yet it is frequent also in high-pressure cases (hyperpiesia) in which by ordinary clinical or pathological methods no renal disease may be detected; (*f*) that indeed it is rather in the low-pressure kind that the general withering with interstitial substitution all over the body takes place, as it does in the kidney; (*g*) that, as a matter of clinical and pathological experience, in the high-pressure cases, including those of renal disease, the heart's muscle, save for consequential strains, largely retains the characters and resources of health, while not infrequently its coronary arteries are practically efficient.

Now, do not these differences suffice to prove that the conceptions which by Huchard's eloquence have held the field so long

must be reconsidered and recast? What are we to think of this sentence, recently from the pen of a leading physician writing specially on heart diseases?—"Arteriosclerosis and atheroma may cause fatty and fibrous degeneration of the cardiac walls by obstruction of the coronary arteries, and *at the same time a general rise of the arterial pressure* is common in this complaint" (italics mine). If we have any use at all for the term cardiosclerosis, it should signify rather the so-called "senile" (decrescent) involution, coronary obliteration, and myocardial fibrosis and decay, than the accumulation of stresses against which, in chronic renal disease or in hyperpiesia, the cardio-arterial system, the heart especially, makes a long and vigorous and not inglorious resistance. From his carefully tabulated experience Schabert[1] says of 15 cases of arteriosclerosis without high pressures ("ohne Hypertension"; he admits my distinction) he found some cardiosclerosis in 12. "Cardiosclerosis" then is an omnibus word; it may mean the cardiofibrosis of Dehio, it may mean coronary atheroma, it may mean valvular induration, and so forth; changes which are several, and have no regular correlations.

If the reader accepts my original separation of arteriosclerosis mainly into the hyperpietic and the decrescent (or involutionary) forms, he will perceive that throughout these two classes the behaviour and conditions of the heart are widely different. In Hyperpiesia, as we may see again and again, the heart is for a long time little the worse; it has more work to do, but for a while it meets this demand without even a static dilatation or great hypertrophy. For many weeks at least, in comparatively young persons, perhaps for some months or a year, I cannot tell, the heart may meet a pressure of 180-200 without demonstrable overgrowth. A normal left ventricle can continue to expel its contents against a double aortic pressure, as may be shown by the constancy of the diastolic pressure and of the pulmonary arterial pressure.[2] In trained men the heart learns to meet large, though temporary, fluctuations of output. Under regular exercise it learns quickly to modify its own

[1] Schabert, *Petersb. med. Wochenschr.*, No. 4, 1911; quoted *Deutsche med. Wochenschr.*, March 2, 1911.

[2] Leonard Hill, private letter January 2, 1908, with reference to Schäfer's *Physiology*, vol. ii. p. 151, fig. 91. So also Roy and others.

tone, and to throw out larger quantities, even against the rising pressures of the outset of exertion, before peripheral dilatation takes place; all which degrees of filling it must do in approximately equal times. This competence one observes in the cases, which we see again and again, of high pressures in renal disease of unknown, but not very long, duration without appreciable cardiac hypertrophy; though, on the other hand, it is true that in urgent renal disease the left ventricle is capable of considerable hypertrophy in a few weeks. A comparatively young heart however, under a correspondingly high-pressure coronary circulation, seems by its reserves to be able for a while to keep up the balance without obvious statical increase; so that if the resistance be reduced, things may and do fall back into their normal relations. The same is true also of moderate degrees even of static hypertrophy, if of no very long standing, of standing not sufficient to have strained the vessels and established in these structures a new set. We see such hearts come back within their normal limits, and the disorder clear up; prone as it may be, if not prevented, to return. If this strain has taken place, if under excessive and persistent arterial pressures irremediable alteration of both heart and arteries has become established, and restoration to the normal hopeless, if indeed dilatation of the heart under the irresistible pressures is now ominous of defeat, and the patient is falling into the abyss of "heart failure," even then the heart may die —so to speak—sword in hand, die bravely and valiantly before superior forces, without intrinsic infirmity. The myocardium in such a case will probably show some fibrosis, and the coronary arteries the effects of strain; but the heart is one which has succumbed hale and fighting; and in subjects under 50 years of age with permeable coronaries the myocardium often proves to be practically sound. Dr. Janeway has aptly pointed out that in these hearts auricular fibrillation is rare.[1] Moreover in high-pressure experiments on animals these enlarged hearts consume a proportionate increase of oxygen; whereas the chemistry of a degenerate heart is degraded, it contains traces of lactic acid and aldehyd, and considerable quantities of amino-acids (Rohde, Ogawa and others).

[1] Janeway, *Arch. Inst. Med.*, Dec. 1913.

But in the decrescent form of arteriosclerosis the story is otherwise; in this series of changes the heart may indeed be involved in a common fate with its vascular extensions. More or less, as accidents may fall out, if it is suffering it is not from overwork, for in these cases the arterial pressures are not excessive, but from a more or less progressive and insidious intrinsic decay. If, enfeebled by degeneration, loss of elasticity, stiffness and tortuosity, and the formation of vortices or otherwise, the arteries offer more frictional resistance, this hindrance does not attain to large proportions; the pressures rise a little, and velocity falls a little; the heart may perhaps undergo some slight increase in volume, but the whole arterial system is put under no extraordinary strain. What happens is that the individual does less work; and thus, under economical conditions, he may live long enough, if less effectually. Not infrequently, in a slow case, as life advances, and the quality of the arteries becomes gradually a little worse, the heart does not deteriorate *pari passu* with the vessels, but takes upon itself a little more work, and does it quietly. Thus by a gradual readjustment of conditions a moderate accumulation of stresses is supportable, and life goes on fairly well. Let us not then be too ready, even in old people, to talk thoughtlessly of "cardiosclerosis" as a part of arteriosclerosis.

Morbid Anatomy of the Heart in Arteriosclerosis.—In discussing the arteries in this context we saw that it was not always easy to discriminate differences of cause, if any, by their anatomical changes. In respect of the heart however some such discrimination is more practicable. I have said that the heart in the series I have called Hyperpiesia differs from the heart in the course of that kind of arteriosclerosis which I have indicated by the name of Decrescent. As in all fields of pathology, transition cases occur to blur or to complicate the description, but here not to any very confusing degrees.

How comes it then that in discussions of Arteriosclerosis we are so familiar with the name "Cardiosclerosis," a sound too often taken for sense? Why, because it is one of those comfortable words which dispense with thought. I have tried to extract, especially from our brilliant French colleagues, some pathological definition, or at least some notion of its meaning as

a name, but in vain. At length however, in an essay by a leading physician of the French school, I lighted upon a definition; it ran thus: "Cardiosclerosis signifies all those particular states of the myocardium which lead to cardiac insufficiency—say in Bright's disease, in diffuse arteriosclerosis, or in states of high tension; in a word, all final stages of the 'cardiopathies artérielles' of Huchard." That is to say, the name is as destitute of precise meaning as I had been led by the indiscriminate use of it to suppose. To offer us in exchange for it Huchard's "cardiopathies artérielles" is to palm off on us one token for another, neither of which has any intrinsic value. Huchard and his disciples reiterated again and again that high tension, cardiosclerosis, and arteriosclerosis were features of one uniform and consistent process.

But from the facts we have learned, not that arteriosclerosis is a "disease," or clinical series, involving heart and arteries in a fairly uniform process, in a common decay; nor again that it is a cause of "high tension" bringing with it, more or less inevitably, strain and destruction of the cardioarterial machine; they tell us that we are dealing with, and confounding together, two different processes, the one a process of mechanical strain with consecutive lesion and destruction, the other a process degenerative from its beginning, whether rooted in some original defect in these parts, implied in prolonged wear and tear, or engendered by some poison or crooked chemistry of the body. From a pathological standpoint however there is something more to be said.

I have argued that in the arteriosclerosis of "high tension"—my Hyperpiesia—the heart primarily is, and throughout the readaptive stages of the malady continues to be, healthy. Hypertrophied it is, but so long as by natural growth the extraordinary work is done, hypertrophy is not disease. The proverbial blacksmith's arm is not a diseased arm, nor does it naturally carry in it the seeds of disease. There is more stuff to nourish, and so far if, by arterial disease or other defect or poison, the fuller tale of wholesome food should fail, the arm is more liable to dwindle. But these are conditions not essential but consequential. I must be forgiven if I reiterate that a man may fall into hyperpiesia, and continue in it for some time, perhaps for a few years—three or four?—so that

his heart, contending against abatements of velocity, may wax in sound fibre to degrees of considerable hypertrophy, and yet, toxæmia apart,[1] remain healthy (p. 9); and, if normal pressures can be restored, may still be capable of returning to its normal work and dimensions. In hyperpiesia neither heart nor vessels are to blame, they are not the sinners but the sinned against; and the heart if defeated falls in a defence of the citadel. It may fall, as I have said, sword in hand, or it may be reduced by the plagues incident to beleaguered communities; in this case, by strain, or by incidental toxins, the gallant heart and its auxiliaries may be compelled to surrender; but this is not degeneracy. Accordingly, if by some chance a man die in an early stage of hyperpiesia, or likewise of chronic renal disease, the left ventricle of the heart will be found hypertrophied, but healthy; at later stages the right side of the heart may suffer a consecutive strain, and, whether by strain alone or by toxic influences arising out of disturbed nutrition, both ventricles may now show evidences of injury, often in chronic cases by the substitution of a worse for a better fibre. As Dr. Batty Shaw well says,[2] "The case is a cardiac case only in a secondary sense, and the heart may be normal in cellular detail." Furthermore, the coronary arteries may not yet have suffered much; to the naked eye they may remain fair, or present patches of decay of little importance in respect of a supply of a due quantity of blood, be it better or worse. Barcroft and Dixon have shown that the consumption of oxygen per unit of heart is much less than per unit of kidney, pancreas, or submaxillary gland; and Bollinger and other pathologists now admit that often in these big overworked hearts no abnormal structure is to be found. As after a single nephrotomy the other kidney enlarges, and with it its arterial trunk, so the heart, and even its arteries, grow up to the new demands; but this is not degeneration. In the last stage of the malady, it is true, the heart will be found big and distorted, stretched beyond its elastic limits, deteriorated in fibre, and, it may be, with its nutritive arteries damaged by overwork so gravely that latterly their inelastic walls and narrower channels had become unable to supply a sufficiency of food to the myocardium. Thus the cheaper fibre may have

[1] Davy, R., *Lt.*, June 22, 1912. [2] Shaw, B., *Clin. J.*, Mar. 27, 1907.

been laid down in the place of some of the active muscle, or perhaps in support of it; or the muscle may in part have succumbed. The valves likewise are pretty sure, in a like fibroid substitution, and a like loss of elasticity, to exhibit the effects of strain. Notwithstanding, even then the heart does not present the typical state due to a primary atheroma, or primary degeneration; in a collection of appropriate specimens it is easy to separate the "high tension" hearts from those which were primarily atheromatous in vessel or degenerate in tissue; those which fought a good fight to the last from those which, by their own inherent imperfection, or perverted by some poison, had deteriorated primarily.

Nevertheless it is true that in many of these hearts, particularly in those of chronic hyperpiesia or renal disease, and when dilatation has supervened, some alteration of histological constituents is to be found, at death perhaps in most of them. The hypertrophy seems to consist, as Dr. Gutch demonstrated in our laboratories, and as Kölliker taught beforetime, rather in an enlargement of the several muscular fibres than in a multiplication of them; frequently however additional fibres are to be seen, even in abundance, not of muscle but of connective tissue. This "*myofibrosis*," often very improperly called myocarditis, was perhaps fully studied first by Dehio;[1] he showed that if the patient had died during a stage of adequate compensation, with but little dilatation, the myofibrosis was nominal in degree; but that with the fag of the heart and its chronic dilatation, the connective element increased. Dehio thought the immediate cause to be viscosity of the nutritive blood. In mitral regurgitation the myofibrosis is found more in the left ventricle, in mitral stenosis in the right ventricle; but it is always most in the auricles. It may be found in hearts with quite normal coronary arteries, as in valvular disease; though in other cases disease of these vessels may have conduced to its formation. As I have asked already: When is fibrosis an effect?—when a cause? In this myofibrosis the connective fibres are sometimes uniformly distributed, like an infiltration penetrating among the muscular elements, some of which may be degenerated (vacuolar); sometimes they are

[1] Dehio, *Deutsche Arch. f. klin. Med.* Bd. lxii., 21 Dec. 1899; also an extract from Dehio, *Petersb. med. Wochenschr.* in Epit. *Brit. Med. Journ.* No. 9, 1901.

patchy in distribution, around the insertions of the valves, in the papillary processes, about the twigs of the myocardial vessels; sometimes diffuse, sometimes in well-defined strands. The diffuse and the patchy forms seem to be of somewhat different nature and origin; but neither of them, unless very extensive, or invading cardinal parts such as valves or conductive bundles, is of much practical importance. It must be difficult in a syncytium such as the myocardium to follow these interlacing strands in their early development; but Fahr, the Prosector of the Hamburg Hospital, in a very interesting paper [1] argues that, as age advances, the muscle of the heart becomes more and more unready to open quickly, and that for this purpose a diffusely arranged elastic network is developed ("die Muskelfibrillen zu umspinnen") as auxiliary in the expansion of the heart in diastole, as in elderly persons the arteries begin to stand open; this material, he says, is largely increased, for the same reason, when the heart is under stress, "as in arteriosclerosis." Jores also says that it is a structural support. However this may be, as Krause says,[2] "In children's (hearts) fibrous tissue is scanty, but increases extraordinarily in adults." This we have seen in the homologous arterial tree; not till about the age of 30 is connective tissue apparent, with Van Giesen's stain, in the intima, and it reaches no considerable development until the decade 40-50. Hirschfelder again (2nd ed. p. 313) regards the gradual increase of connective fibre as a strengthening rather than a weakening of the chambers of the heart. This fibrosis may lie on the marches between physiology and pathology; but Fahr also points out that only in the new-born infant does the myocardium consist of pure unsupported muscular fibres; that then a fine connective network begins to embrace the muscular fibres, and increases as the heart has more and more to do. Thus it lies parallel, as it were, with Thoma's and Jores' regenerative fibrosis, and such a name as "endocard*itis* fibrosa" is inapplicable. Tripier,[3] in refinements which are beyond me, still regards these fibrous changes as "inflammatory," but he seems to observe no distinction between inflammation and

[1] Fahr, *Virch. Arch.*, 1906.
[2] Krause, *Normale Histologie*, 1911, p. 208.
[3] Tripier, *Études anat.-clin.*, 1909.

active hypertrophy. I still hold to my postulate of fifty years ago, that inflammation implies lesion and repair of lesion; arteriosclerosis is lesion, but the repair is insignificant. How again are we to accept the statement of a distinguished recent French author who says that a "sclerous *myocarditis*" is one of the manifestations of arteriosclerosis? In England,[1] France, and Germany the best observers nearly all agree that the process, if a proliferative reaction, is not inflammatory; often it starts not around the interstitial vessels but remotely from them, whither nutrition has less instant access. Marks of old infection, such as the rheumatic or the diphtheritic, have other characters; the fibrosis is perivasal, the vessels themselves are thickened at the foci, and small scars testify to the bygone mischief. From the present category then chronic myocarditis is excluded. Krehl and other pathologists tell us that elastic fibre also begins to appear in the maturing heart, and increases up to old age; but connective fibre has about 100 times the resistance to stretch of elastic fibre.

Once more, Aschoff says decisively (*loc. cit.* ii. 37) that it is false to regard the so-called "myofibrosis" as inflammatory or a sign of weakening of muscular energy ("Muskelkraft"). He calls it "compensatory"; and if it be so, or at any rate auxiliary, the process must be distinguished in reason from an irritative or atrophic fibrosis (p. 15). If this argument be sound, and the facts verified, to name this change "Chronic sclerous myocarditis" is as wrong as Cardiosclerosis is misleading. Dr. Cowan[2] however describes three kinds of cardiac fibrosis: (1) Periarterial fibrosis, never extensive; (2) fibrosis in much larger patches, remote from vessels (dystrophic); (3) large fibrous lumps (callus). The first kind he attributes to a reaction to various causes, such as infections. Hypertrophied hearts, he says, are not necessarily fibrous but frequently are so, more or less. In the periarterial form Dr. Aldren Wright (*loc. cit.*) found about the small twigs fine fibrillary connective tissue in a ground substance, and numerous connective corpuscles, flat spindle or irregular nucleated branching cells, with long axes parallel to the vessel.

[1] Cf. Mackenzie, *Brit. Med. Journ.*, Oct. 20, 1906.
[2] Cowan, J., *Journ. of Path. and Bact.*, Dec. 1903.

Dr. J. Mackenzie takes a different view, saying that myocardial fibrosis is in inverse proportion to the heart's capacity; but he gives no histological proofs of this. I have sometimes guessed that, old hearts being drier, their electro-conductivity may be less. The arteriosclerotic kidney gets on well enough (p. 371), but in each and every part, whatever the reason and however normal the myocardium may seem, an old man has a narrower reserve. Brault, when he considered the nodes and trabeculæ of connective fibre in these hearts as insignificant, as mere curiosities, went perhaps to the opposite extreme. However, his testimony may be added to mine, that in not a few hypertrophied hearts, even of Bright's Disease, the myocardium—unless at a few small points of stress—is histologically normal; the striations are clear and the nuclei well stained. But in the later stages, if no nodules be present, streaks of connective fibre traverse and interpenetrate the myocardium. These changes, and the nodules also, are commonly found in the hearts of aortic regurgitation; and again of atherosclerosis without high pressures.

In many fibrotic hearts the muscular fibres, although embedded in this fibre, are often demonstrably quite healthy, even in the papillary muscles. On the other hand they may be as distinctly diseased, and heart failure on the way. Thus in the heart of an old man, the subject of arteriosclerosis, recently examined in our laboratories by Dr. Haynes, although the ample coronary trunks admitted a large probe freely (and we know from Jores that in these cases of coronary atheroma the interstitial vascular network is usually intact), with some myocardial fibrosis the muscular fibres were widely degenerated. In some hearts of long and good service there is plenty of fibrosis; in others, which had proved rather decrepit, and prematurely so, whatever the other marks of muscular decay fibrosis is scanty.

For a considerable degree of interstitial fibrosis a long time seems necessary; it signifies a heart long under some disadvantage; we find it not in old hearts only but in younger hearts if crippled by old-standing valvular defect. It seems proportional to strain, and apparently offers resistance to it.[1] In

[1] See Stadler, *Deutsche Arch. f. klin. Med.*, 1907.

the auricles fibrosis is often almost complete, although the auricular branches of the coronaries may be but little diseased.

As in the arteries then so in the myocardium, the effect of laying down this inferior fibre serves in part to support the muscle by a tougher tissue, but at the expense of the limits of elasticity; resistance is gained at the price of less resilience; once overstretched, the contractile organ will be the less able to return to its normal diameter. The auricles, when so converted, may almost cease to be contractile sacs. It is not impossible however that by its native tendency to contract it may have some bracing effect upon the fabric.

In some paragraphs on cardiosclerosis in his work on the Heart (pp. 237-8), Dr. James Mackenzie identifies this term with fibroid heart, but he scarcely distinguishes between interstitial fibrosis, which proceeds from childhood to old age, and "replacement fibrosis"; indeed, he rather assumes that all cardiac-fibrosis is substitutive. He then marvels, as well he may, on the diversity of symptoms in patients in whom this alteration is found after death. In many cases the alteration of the heart had little or nothing to do with the symptoms—as *e.g.* his cases of angina pectoris; in others the relation was but correlation, was but the fibrosis which in advancing years is general in man, and throughout the bodily tissues. The cardiac fibrosis may have had as little to do with the symptoms of a particular case as the fibrosis of the skin or of the pancreas. Dr. Mackenzie dwells with much valuable exposition on limited cardiac response, but here again one desiderates more distinction between the direct cardiac lability of disease and that lessened cardiovascular capacity which, waning as "The Strong Hours work their Will," is still compatible with longevity. And in high blood pressure, on the other hand, the so-called "degenerate sclerotic heart" is often sustaining a blood pressure, of systole and diastole, of 20 per cent or more above normal.

Dr. Mackenzie says, after Huchard, that persistently excessive pressures are "necessary" for the production of well-marked "cardiosclerosis." If so, we must contrast this cardiac fibrosis of high pressures ("high tension")—a fibrosis of defence—with another fibrosis, more dystrophic in nature, of decrescent arteriosclerosis; though the difference may be rather inferential than

histological. The first would be the "compensatory" process; the others degradations, gradual or static, cheaper products or remnants, or disseminate sequels of toxins such as those of diphtheria or rheumatic fever. If then the name cardiosclerosis is to be used at all, it must be given to fibrosis the result of coronary atheroma or senile withering, without high pressures or other stress upon the heart.

Dilatation is at least of *two kinds*: that which is an adaptation for a larger mass of blood (contraction-volume)—whether relatively to the other chambers or absolutely in respect of the whole system—and the yielding under distention, due to disease or only to loss of tone. We cannot admit with certain Leipzig pathologists that a dilated heart always signifies failure—failure by loss of nutrition, or by myocardial infection, or even by loss of tone; clinical experience is against this assumption: we meet with many a heart both hypertrophied and dilated yet in functional equilibrium; and conversely we can rely on no direct relation between dilatation and morbid histology.[1] Myofibrosis and hypertrophied muscular fibres may be co-ordinate. Aschoff and Tawara examined 150 hearts consecutively, on Krehl's method, and found no correlation between excess of connective tissue and failing hypertrophy (p. 11). Indeed we have no anatomical facts to show where and how in these cases dynamic default becomes manifest statically as structural change. Aschoff and Tawara think that, apart from increasing valvular incompetency, hypertrophied hearts fail because of minute scattered "Schwiele" diffused under the endocardium by thrombo-endocarditis. The "Schwiele," in its mature and permanent state a scar, consists at first in a focus of congestion in which grow fibroblasts and phagocytes; this focus quickly becomes anæmic, or, if in a district of coronary circulation without ready anastomosis, is from the outset anæmic, and a white scar or callus, consisting of fibrous tissue, is formed (*Cicatrix myocardii*). These bodies may, as we know, reach very large dimensions. Generally speaking, they are of thrombotic or embolic origin; and if they form, as often they do, in the columnar part of the ventricle the apex contraction is impaired

[1] See Myocardial Values, p. 32.

and the mitral flaps are slackened or, ultimately, distorted.[1] Bäumler [2] states that when in lumps they are due to coronary disease or chronic nephritis, but when in broader and flatter breadths they are old masses of toxic origin.

To turn to my own experience, this kind of fibrosis, unless in considerable quantity, is not directly related to heart failure (p. 11). In its beginning it is most easily found about the apex and the columnar region, and in the septum. In its earlier stages it appears as yellowish moist streaks—Ziegler's "soft sclerosis"—then, as it contracts and withers, it becomes the more or less pearly scar with raylike extensions into the neighbouring myocardium. If these coalesce, large callus is formed, with strands of muscle relics lying between the several primary spots. Cohnheim's well-known experiment may here be recalled to mind in which he clamped out portions of the ventricular wall without any consequent fall of arterial pressure. The heart of a healthy old man may be larger and seem stronger than that of a weedy youth; whether under various kinds of stress it is essentially more enduring is another matter. With increasing years the heart often increases a little in bulk; and, as we know, the systolic pressures gradually rise to a moderate excess—say to 140, or even 150. The diffuse form of fibrosis, in fine lines or patches, often first laid down in the mitral columns, is in these larger hearts more abundant, and the walls are rather thicker and a little harder to the knife; but this, as we have seen at length, is not "myocarditis." Indeed *this kind of change, even if widespread, "is usually a post-mortem finding" (Josué); it gives rise to no symptoms, nor does the patient die of it.* Josué says truly that by this fibrosis, so long as it does not affect the valve mechanism or conductive tracts, cardiac function is little impaired. There is no consistency in its degree, nor even in its concurrence, with symptoms of failure. This fibrosis is often a part of a universal change which age brings with it, not only throughout the arterial tree, but also throughout all senile structures. We have followed this fibrosis in deteriorating arteries and seen that it may lie between normal elastic layers. The elastica may, of course, split

[1] Aschoff, *Lehrbuch P.A.*, 1909, Bd. ii. pp. 18 and 35.
[2] Bäumler, *Deutsche Arch. f. klin. Med.* Bd. ciii., 1911.

independently, and nodular atheroma is prone to impose itself upon the diffuser product and, becoming confluent, to involve the whole vessel in decay. But, atheroma apart, we have no evidence that the change in later life, whether in heart or vessel, is one of much gravity. The whole tree is, of course, less resilient, with the consequences we have discussed.

A little cardiac enlargement then may be a character of decrescent arteriosclerosis, though in this condition, if uncomplicated, the heart never attains anything like the dimensions of that of hyperpiesia or of chronic renal disease. To the eye and hand the organ is solid rather than big. Charcot, in 36 cases of arteriosclerosis without valvular disease, found the heart somewhat enlarged in 36 per cent; and in the Salpetrière it is probable that the large majority of cases were of the decrescent kind.

We have dealt with the assumption in current writing, though particular writers may here or there modify their statements, that arteriosclerosis of whatsoever kind, or of whatsoever clinical process, is the cause of cardiac hypertrophy. Bourguignon [1] wonders how "old sclerosed" hearts can stand for years excesses of blood pressure, sometimes double the normal. They don't. The heart of high pressure may be sound till near the end. Because arteriosclerosis is found it does not follow that the pressures had been high. For the heart to grow under high pressures is one thing; to be reduced in quality by sclerosing coronaries is another. This Huchard and his pupils did not see. But we have seen that in hyperpiesia the cardiac hypertrophy is not a result of the arteriosclerosis, which is a subsequent event, but of the peripheral resistance; yet the assumption still prevails that in all and any arteriosclerosis it is this change which augments, directly, the dimensions of the heart, an inveterate opinion which has been fully criticised in preceding pages. If in a body presenting senile arteriosclerosis, even to grotesque degrees, the heart be increased at all, the increase is quite moderate, such as during life could not be detected by physical diagnosis. Here again we are beset by the notion of "provisions of nature"; because more work might be expedient to overcome failing storage of energy in the vessels, therefore such more work is provided.

[1] Bourguignon, French Congress of Med., Geneva, Sept. 1908.

If provided at all, it is in quite subordinate measure; indeed if considerable it might force the failing vessels. The consequence in elderly arteriosclerotics is not so much a circulation compensated up to or near the normal values of younger and sounder persons, as in a moderate aortic regurgitation for instance, but a narrower range of effort and perseverance. Elderly persons live the quieter life that their age and their languor dictate; their blood runs more slowly. An attempt, if foolishly made, to vie with the livelier habits of youth, would lead not to the accelerative elastic capacity and output possible to the younger heart, but to arhythmia and incomplete emptying of the chambers. The heart of the old man cannot do it. Whatsoever dissipation of energy there may be is not compensated, neither persistently nor temporarily; the automatic energy of the myocardium is less; moreover if its own arteries are implicated in the same degeneration, they are worse carriers, and the functions of each and every organ and part are less exacting.

We have seen then that, histologically speaking, fibrosis and inefficiency are by no means parallel; we are not at all sure that some diffuse fibrosis is a change of much gravity (p. 49); it may indeed be conservative. We have found it difficult to discriminate between such *a fibrosis, apparently conservative, and a mere dystrophic sclerosis* such as may be found more or less throughout the structures of an old man. This general sclerosis can scarcely be conservative, it is simply cheap. To talk of a "fibroid diathesis" is mere verbiage. In the heart, as elsewhere, dystrophic sclerosis usually depends on defect of the nutrient vessels; it is a "substitution fibrosis," not dependent on any exudative or periarteritic irritation.[1] Very slow deterioration of the coronary branches, if protracted over years, may lead to a substitution fibrosis, as on the other hand prolonged stretching, within limits, leads to the conservative fibrosis: a fibrosis, as Councilman and others have demonstrated, in hearts whose coronary arteries were not only efficient, but, as is common in hypertrophy, themselves also enlarged. Dr. Shinglewood Taylor, in a thesis on Aortic Stenosis for our M.D. degree (1914), was "surprised in many cases to find extensive

[1] For a discussion of this kind of fibrosis see Adami's *Pathology*, vol. i. (2nd ed. 1910).

fibrosis of the left ventricle with no apparent loss of efficiency." With increasing work the coronaries frequently enlarge. With the sustenance of the heart after closure of the coronary arteries, or their mouths, I shall deal later (pp. 23 and 363).

Gennari [1] set himself to compare the somewhat contradictory statements of Krehl and of Dehio, on the one hand, that myofibrosis is constant in hypertrophic states of the myocardium and may be one cause of its ultimate default, and on the other, of Aschoff and Bollinger, and I may add of Kanthack and myself, that, on the contrary, this alteration is often absent. Gennari took nine hypertrophied hearts, selecting such as to the naked eye presented no lesion. Of these he made many and various sections, and in two of them, after the most prolonged search, no fibrotic invasion could be detected. In the remaining cases fibrosis was found, but very irregularly; sometimes only in a small proportion of the sections examined. Moreover, in apparent date, the fibrotic areas bore no relation to the phases of the clinical symptoms. Indeed, in perusing his paper, I inferred that in some of them the fibrotic areas described were evidently relics of some long-past infection. On the whole Gennari concluded that the secret of myocardial failure did not lie in fibrosis, whether inflammatory, compensatory, or substitutive. Bradycardia, which formerly was attributed to the kind of this ventricular deterioration, is now explained by the seat of it.

I suggest then, if conjecture in pathology may be forgiven, that a distinction should be made between auxiliary and substitutive fibrosis, and that the distinction may be this: that whereas in substitutive fibrosis the genuine muscular fibres are supplanted by the fibrous, in the auxiliary mode a substantial proportion of the fibrous tissue may be laid down in support of the muscular. If we may suppose, on the comparison I have made more than once, that by arduous work juices are squeezed out into the myocardium after the manner of the juices on the lee-side of a tree, so in the myocardium, expressed juices may be converted into, or feed, either a larger bulk of muscle or of muscle and connective fibre, as the result may be. The one issue is muscular hypertrophy; the other is a myo-

[1] Gennari, *Arch. per le sci. med.*, Dec. 1905.

fibrosis, yet still of a conservative mode, of a mode auxiliary to, if not the full equivalent of, the "nobler" fibre. It is the second best. The third best or substitutive mode arises, probably, out of failing capacity of the nutritive factors to sustain the "nobler" standard; muscle gives way to a cheaper and inferior kind of fibre. I have seen it stated in works on gynæcology that if a fibroid tumour pull upon a long stalk, or if its vessels are interfered with by the pressure of surrounding parts, its muscular fibre is so far supplanted by connective tissue. Thus, if a ventricle of the heart be submitted to dilating stresses, the extra juices thrown out may make more muscle or, under less favourable conditions whether of stress or of nutrition, muscle with a buttress of connective fibre; both processes being conservative. If however dilating stresses be excessive and nutrition fall short of the best, then no addition may be made to the muscle, but the normal scaffolding of connective fibre may be fortified; if nutritive capacity be still more in default, connective fibre will be the utmost that the impaired capacity of the self-regulating machine can produce, the muscle itself will be supplanted by the lower fibre. If so, the distinction between supplementary and substitutive myofibrosis is real. The one, let us say, in a ventricle submitted to dilating stresses, is an adaptation and relatively an advantage; the other is simply tissue degradation in a ventricle perhaps exposed to no excessive stress, presenting it may be no dilatation, but falling short of its own proper structure. Such a conjecture would, I think, reconcile the diverse opinions of sundry writers, such as of Krehl and Dehio of the one part, and of Aschoff and Bollinger, Kanthack and myself, of the other. It is almost needless to add that in the same heart both kinds of fibrosis—the protective and the substitutive—may be mingled in incalculable degrees; as, for instance, in an old and slow case of aortic regurgitation with hypertrophy, dilatation, and atheromatous coronary arteries. In an extremer degradation of the myocardium, not even connective fibre can be kept up, and the muscle falls into granular or vacuolar demolition. Cardiosclerosis then, unless used simply and clearly to mean myofibrosis, is but a vague term, and in any case is not wanted.

To go back to the beginning; these considerations emphasise

the fallacy of the confusion of hyperpietic and decrescent arteriosclerosis, as in Huchard's (*passim*) three stages of "cardiosclerosis"; firstly, the "presclerotic," with high tension; secondly, the stage of cardiac degeneration (cardiosclerosis proper); thirdly, the "mitro-arterial." In the first stage, he said, "arterial lesions are a minimum," and are curable; in my interpretation arterial lesions in this stage have not begun, or are only nominal, they are consequential, and often tardy; the third stage he regarded, not as one of a defeated heart, frequently hypertrophied and with patent coronaries, and presenting only auxiliary fibrosis, but as one of an intrinsic decay, not consequential but primary; a part of a general toxic cardioarterial deterioration. Such a series we may find in my Decrescent mode; but in Hyperpiesia, or chronic renal disease, there is no such intrinsic decay, but a perpetual mechanical strain resisted better or worse according to the original quality of the individual system and its reciprocating organs.

Again, in respect of the third or "mitro-arterial" stage, Huchard and his school fail to draw the intimate distinction between the mitral insufficiency caused mechanically by high tension with dilatation of the wall of the chamber, and atherosclerotic decay advancing from the aortic area to the anterior curtain of the mitral valve. The former series—in Hyperpiesia or Renal disease—usually issues in the ordinary course of "big dilated heart," with scanty lateritious urine, tender liver, cardiac dyspnea, and dropsy; the latter series in gradual decay—"cardiac failure," without these overt and unequivocal symptoms and signs of rising venous pressures and cardiac conflict.

We have to consider further the kind of so-called "cardiosclerosis" which I postponed (p. 15); namely, the formation of *callosities or nodules in the heart*, masses regarded by many writers as fraught with grave mischief as impediments to the ventricular contractions. To the minuter and more gritty form of these nodules and the remnants of old infections I have already alluded. On the larger masses an important early essay was written by Thiele more than twenty years ago;[1] but many years

[1] Thiele, *Über Herzschwielen mit Veränderungen der Coronärarterien*, I.D., Göttingen, 1892.

before that Hilton Fagge,[1] and after him Gowers,[2] and Charlwood Turner,[3] had led the way; and six years after them the late Lindsay Steven [4] followed up the enquiry. Dr. Turner attributed the nodules to defects of the coronary arteries. Karl Huber [5] had detected the part of the coronaries in what he called erroneously —as we now know (p. 11)—"chronic myocarditis," and demonstrated these changes in 18 cases. In the early papers callus formation was not clearly distinguished from the diffused forms of myofibrosis, auxiliary or atrophic, which we have discussed already; but Lindsay Steven rightly argued that these callosities, often as large as a split pea or an almond, or still larger, should not be classed either with chronic myocarditis or diffuse fibrosis.

The coronary artery leading to the patch may however be healthy; in this case the callus may be attributable to infarction; embolus being far less frequent than thrombus. An infarct may be followed by a hæmorrhagic exudation, and this, "becoming organised," may contribute its part to the mass. Or smaller patches may coalesce to make up a large callus. If a coronary plug be very big and sudden, more grievous symptoms or death may ensue: these cases I shall deal with in my chapter on Angina Pectoris, p. 450. Masses thus produced may lie deep in the heart substance, or may be subendocardial or subpericardial; they are most frequent in the left ventricle, and towards the apex, where the well-known ectasy and rupture may occur (*aneurysma cordis*).

As regards the dependence of fibrosis, nodular or diffuse, upon the coronary circulation, there are many facts to check too ready assumptions on the subject. Among the hearts which Kanthack examined with me, I remember one in particular which was notable in this respect, that not only were the coronary arteries calcified, but their orifices also were so utterly obliterated that the very seat of them was indefinable; yet in this heart, so far as the microscope could tell us, the myocardium was normal. And this experience is far from singular; a greater or less abatement of the coronary circulation with, or even without, comparatively slight degrees of fibrosis is not uncommon

[1] Fagge, H., *Trans. Path. Soc.*, London, vol. xxv. 1874.
[2] Gowers, W. R., *Reynolds' System*, 1877, vol. iv.
[3] Charlwood Turner, *Trans. Med. Congress*, London, 1881.
[4] Steven, L., *Lancet*, Dec. 1887. [5] Huber, K., *Virch. Arch.*, 1882.

(p. 267). Létulle, by two examples especially, enforced the maxim that coronary occlusion need not imply myocardial decay, but that "even considerable hypertrophy of the heart may coexist with coronaries obliterated by atheroma." Bäumler,[1] in a careful study of the subject, questions the invariable dependence of callus upon coronary disease or infarction; he believes that in many an instance he has demonstrated these knots in parts of the myocardium where the respective coronary branch was pervious, and he quotes Gierke and Ziegler to the same effect. Most pathologists indeed, as Fahr of Hamburg[2] for instance, now admit that, often as callus and coronary stenosis or occlusion are associated, yet they stand in no direct proportion one to another—in no parallel. If with extreme coronary disease some hearts present much callus, others show but little; and conversely some with much callus show little coronary obsolescence. Bäumler throws the scars of bygone infective foci into the same class, where they seem to me rather out of place; if within themselves they may pursue a similar histological course, their history and significance are otherwise.

It may not be altogether out of place to quote here Bäumler's corresponding notes on the clinical signs, and the symptoms by which he thinks the presence of these knots may be betrayed: these are, a feeble first sound and a loss of the apex beat, independently established; for instance, that digitalis may bring back the apex beat without restoring the quality of the first sound. If, however, he adds, the fibroid thickening is much towards the apex and the heart is slack, the blood charge (contraction volume) may force itself against the chest wall; especially if a cardiac aneurysm be forming there. Sir William Gowers, in a pioneer article on Fibroid Heart (*loc. cit.*), had previously pointed out the signs of defective apex beat and impaired first sound; but it seems to me that these signs, however truly a result of callus formation in the ventricle, are too universal in enfeebled hearts to be admitted as criterions of callus formation, corroborative as they may be in conjunction with other indications.

One important factor in the *comparative immunity of the myocardium under deprivation of coronary circulation* is time;

[1] Bäumler, *Deutsche Arch. f. klin. Med.*, 1911.
[2] Fahr, *Virch. Arch.*, Feb. 1906.

if the process of obliteration be very gradual, alternative supplies of nutriment can somehow be contributed (*vide* Angina Pect., pp. 363 *et seq.*). Every one who has worked with the perfused heart knows upon how little—a few calcium ions, etc.—it can live and work for a while. One alternative access may be by the veins of Thebesius; by their subsidiary aid, in spite of coronary sclerosis, nutrition may be kept up; so that it will not do from the state of these vessels to infer a decayed myocardium (see Morison, p. 362). Coronary sclerosis and myofibrosis do not run parallel. On the surrogation of the veins of Thebesius some interesting researches were made by Porter,[1] and by Pratt[2] of Harvard. Pratt found that if in the excised heart (of the cat or dog), while the heart is kept beating with defibrinated blood (in this experiment Ringer's solution is not sufficient), the coronary arteries be closed, and then one of the coronary veins incised, a slight but continuous stream of blood will flow from the interior of the ventricle through the foramina of Thebesius into the coronary veins, and out at the artificial opening; moreover, in its course through the heart walls it becomes venous (see Redwitz, p. 364). Wardrop[3] had made some interesting remarks to the same effect; and he in his turn quoted some corroboration from Abernethy.[4] Jores' recent demonstration that atherosclerosis, which so readily attacks the coronary trunks and branches, does not readily invade their finer ramifications,[5] and the free inosculations demonstrated by West and by Spalteholz (p. 363) make this alternative irrigation, both in experiment and in clinical medicine, more intelligible.

Now let us turn from the myocardium to the inside of the heart, and especially to the valves; there sclerosis—cardiosclerosis if you please—may be conspicuous. Of sclerotic disease in and about the aortic valve we are abundantly informed, but a part of this information is that even the extremer degrees of this lesion are associated not infrequently with a very fair myo-

[1] Porter, *Brit. Assoc.*, Toronto, 1897.

[2] Pratt, *Amer. Journ. Physiol.*, Jan. 1898. See also note p. 49.

[3] Wardrop, *Diseases of Heart*, 1859.

[4] Abernethy, *Philosophical Transactions*, 1798.

[5] See *e.g.* in Jores' 1904 paper in *Virch. Arch.*; the old woman of 71; senile dementia and advanced general arteriosclerosis; the coronary twigs within the heart's muscle, as likewise the twigs in the skeletal muscles, were unaffected.

cardium. Now and again in old atheromatous patients I have seen, on the establishment of aortic regurgitation, the left ventricle, in spite of supposed sclerosis, hypertrophy not a little to meet the new conditions (*e.g.* Vol. I. p. 453). We know also how by the ear in the living patient, and by the eye in the necropsy, the mitral valve is liable, whether of its own frailty under normal or abnormal stress, or by extension from the aortic area, to suffer sclerosis; and indeed that under these conditions a corresponding systolic murmur during life is no very grave item in prognosis.

The common experience that, as years are added to years, the pressure in the arteries normally rises somewhat, must signify fair cardiac efficiency; were the heart, by whatsoever common "sclerotic" or other change, to lose contractile value, the disadvantage would be so multiplied that few of us would cross the threshold of old age. My own impression, a superficial one I admit, is that in such later periods the heart rather waxes than wanes; but to determine this question difficult comparisons both of weight and quality would have to be made.

Finally, in some few cases we may see a very severe and extreme change of the whole heart's muscle, chiefly of the left ventricle, in which the part is almost wholly converted into dense harsh fibrous structure, diffuse knotty and nodular, without any notable dilatation or disease of the coronary vessels. The condition is an obscure one, and in nature different; I note it here only to distinguish it from Dehio's alteration which we find first in the auricles, and from callosities.

Cardiosclerosis, then, is a name signifying no definite conception; on analysis its meanings prove to be divers and confused. Huchard applied it not to the heart of decrescent arteriosclerosis, of which he had no discernment, though to this it might have been given with less ambiguity, but to the heart of hyperpiesis, the big dilated heart overstretched by high tensions (the "Hochdruckstauung" of the Germans), and defeated at length by them; ultimately with a mitral insufficiency, due rather to forcing of the mitral orifice than to sclerosis of the valve. If the name cardiosclerosis be applicable at all, it should denote aortic decay with cognate atheroma of its whole area, of the aortic valve and ring, the aortic cusp of the mitral, and the coronaries; for in the hyperpietic series the

scleroses, if any, are a secondary, a late, and not an essential, feature; indeed the whole heart differs but little from that of chronic renal disease.

Myocardial Values.—In my youth it was a common jest to ask what N. or M. died of, the witty answer being "shortness of breath, of course." The jest has vanished, but the platitude remains, with some alteration of the formula, which, instead of "shortness of breath," now runs "of heart failure." In the obituary notices of our current journals we read daily of these deaths by the half-dozen, that N. or M. died of "heart failure"; and the vogue of appendicitis is threatened. Now, do we owe this new fashion to the physician or to the pathologist? for it is often worth while to observe the straws floating in the wind; we may learn something from some of them. Is this a parrot phrase put up as a kind of shorthand between the doctor and the registrar of deaths? or does it, like appendicitis, signify a fresh point in pathology, claiming its catchword? Does the phrase mean that a snap of the heart stopped the whole machine, otherwise capable of working on, as a snap of a connecting rod may stop the engines of the *Majestic*? or does it mean that the heart was the *ultimum moriens*, and that, so long as this central organ held to it, death was kept at bay? In this case to say that one died of "heart failure" is surely as empty a truism as to say that he died of "shortness of breath." On the other hand, as it is true that we have of late years learned more—much more—of the heart's work and of its diseases, both on the clinical side and on the side of pathology, so the new phrase may be a shadow thrown by some shift of position—whether clinical or pathological—in our view of the heart and its functions. Four or five years ago, when engaged on an article on the treatment of heart disease for Musser and Kelly's *System of Therapeutics*, I was brought closer to this problem and found the answer so difficult that it has occupied much of my attention since, and I have sought the opinions of my friends upon it.

Now, if I appeal to a clinical physician, asking him what he knows about "heart failure," I get a ready answer: I hear of many a case of sudden and mortal stoppage of the heart; of heart failure in infections; of cardiac dilatation and

defeat under high pressures, venous or arterial; of senile decay, so undermining the heart that it can work no more; of angina pectoris; of aortic regurgitation, and so on — his data are in plenty, his opinions decisive. And likewise, when I betake myself to the pathologist, I find him no less ready for me: from him I hear of fatty degeneration; of "cardiosclerosis"—whatsoever this may mean; of rheumatic, diphtheritic, and such-like myocarditis; of fibroid disease of the ventricular walls; of sapping of the *a-v* bundle, and so forth; data again in plenty, opinions decisive. But when I desire to link up the two classes of information, when I confront the physician with the pathologist and seek to correlate these times, periods, and modes of disease and death the one with the other, I find myself but little nearer to practical guidance than I was before. Of course, we all know that a "fatty" heart is not so good as a sound one; we all know that a senile heart will not sustain an attack of influenza like a young one; we all know now that damage of the *a-v* bundle may disconcert the rhythms: but when we ask the physician to draw a parallel in the living patient between the formidable modes of decay described by the pathologist, how and when these degradations are manifested, how, in the long course of cardiac decay, the imminence of sudden death is to be foreseen and provided against; how indeed we are to know that any such process is at work at all; or, lastly, how in a case of known heart disease the degrees of its advancement and of the cardiac reserves are to be noted and tested—then the physician retires from the argument. And so with the pathologist: when we show him a heart from a case of sudden death he will confidently demonstrate to us on the specimen why a heart so diseased could not have gone on; but if beside this one we place another heart—still more diseased, it may be—and tell him that the possessor of this heart lived for long enough, and perhaps in the end died of something else; or if we show him yet another in which, by the poison of rheumatic fever, the tract of Tawara was attacked, and yet, notwithstanding, there was during life no considerable divorce of auricle and ventricle, he in his turn retires from the argument, saying that he is not concerned with clinical conundrums. Thus we have the physician and the pathologist trotting each on his own side of the hedge, each intent upon his

own scouting and his own bearings, and neither able as yet to reconcile his own observations with those of his comrade; both of them being perhaps a little too indifferent to the need of mutual counsel and comparison.

Both Vaquez and Wenkebach at the London International Congress asked (as I had done) how it was that prognosis or even diagnosis in myocardial disease was so unsatisfactory, or even totally erroneous, that we had no measure of cardiac insufficiency. The difficulty lies of course in the complexity of the circulatory coefficients and the academic character of the means of diagnosis of cardiac reserve, means admittedly as yet of little clinical service. Professor Thayer went so far as to say that diagnosis of heart weakness was almost always wrong; and other speakers of experience agreed with him. One of us remarked that the best notion to be got was "the poor one of watching the case throughout"—if there be any "case."

Whether it be then for lack of mutual co-operation, or because of the perplexities of the subject, the interpretation of cardiac pathology in terms of clinical medicine, so far from becoming clearer, has darkened, and the intricacies of the path have become more baffling. Our fathers, knowing less of the entanglements of the subject, and newly provided with a collection of the blunter facts of morbid anatomy, took the matter more easily. That people died suddenly of heart disease was for them an old story; when therefore they were introduced to diseases of the valves of the organ, and to coarse lesions of its substance, their difficulties were almost solved. The patient is dead, here is the lesion which caused his death; what more does one want? But, as pathology became more and more curious, these simple solutions became less and less acceptable. Kirkes, Bence Jones, Wilks, Fagge, T. H. Green, and many others with them, began to demur. In the dead man lesions may have been discovered enough, and apparently fully enough, to cause his death; but the man had died suddenly, and the lesions under demonstration were of long standing, or, in a series of such cases, were very various in seat and nature. Again, in a series of such cases valves were diseased and not diseased; kidneys were diseased and not diseased; atheroma was considerable and inconsiderable; the ventricles were dilated or were not dilated; the myocardium

was in extreme decay or virtually normal; its lesions were now focal, now diffused. Thus gradually it dawned upon the pathologist that the problems of cardiac failure were not so simple as in the rise of morbid anatomy had been supposed; and upon the physician, that his clinical methods were fallible indeed, far more so than had been supposed during the rise of stethoscopy. By what system of compensations was it that the same heart could be at once fairly competent clinically and yet pathologically the seat of old and intimate decay? By what system of compensations could a heart go on fairly well, or indeed without suspicion, while its structure was being undermined? Surely, before the sudden collapse, there ought to have been some stage of manifest incompetency, or at least of falter?

In no organ is this lack of concord between the signals of life and death so disconcerting as in the heart. As Dr. Hector Mackenzie has said,[1] "the heart that failed had sometimes been passed sound in a searching examination for life insurance only a short time before." In all living structures there is a factor of safety, that potential which in the heart we call reserve; but in none perhaps is it so large as in the heart. Moreover this reserve is not wholly muscular; there is the coefficient of arterial elasticity also, and an unknown coefficient of nervous reinforcement. And we may confuse cause and effect; thus excess of blood pressure may not be, as ordinarily, mere constriction, but a lack of depressor response. Hasebroek looks to the degree of arteriolar propulsion, which he says can be exhibited in the rabbit's ear; Broadbent and Rosenbach looked rather to the rhythmical activity of the tissues. Yet of this reserve, of what the heart, in case of need, can do for output, for strength and velocity of systole, we have no scientific measure; the microscopist cannot detect it, the clinician has no valid tests for it. At one time it maintains a defective heart; at another time it betrays a heart which, to all appearance, if not impeccable was at any rate not plainly corrupt. So more thoughtful observers began to put the matter more cautiously, and to say that "when a patient succumbs to heart failure certain degenerative changes are *usually* found in the myocardium and vessels, and these are *supposed to* account for the heart failure." Evidently even yet

[1] Mackenzie, H., *Lancet*, March 23, 1912.

some more refined investigation into the phases and intricacies of cardiac qualities must be instituted if these apparent caprices, clinically speaking, of the heart of man are to be explained, computed, and foreseen. "Heart failure" is evidently a very composite notion—one which must be submitted to a no less penetrating analysis. We are beginning to learn in the heart, as we learn in less striking ways in other forms of organised matter, that complexity of structure and conditions mean not less but more stability; that equilibrium endangered in one direction, or in more than one, may be supported by readaptations of other factors of the concert; so that the organ, if it may not be restored to its pristine capacities, may be kept in some tolerable adequacy, such as, under favourable circumstances and within limits not too wide, to preserve it from default. The heart of an active undergraduate, if slightly and temporarily impaired by a "bad cold," may by the stresses of rowing become gravely harmed, and give rise to unmistakable clinical symptoms; while a senile heart, far more radically vitiated, may for years amble comfortably along the serener paths of old age. *Non intelligitur quando obrepit senectus.*

To pass from these general reflections into more concrete observations, we may begin either with the clinical or the pathological side: as the clinical side is the field of practice, let us begin there; remembering how Goethe taught us that the general is always to be found in the particular. In the clinical field we shall first be impressed by the part of the heart in almost all diseases, and under conditions such as anæsthesia. In infections we know that the heart is concerned in three ways: in some of them it is directly attacked; in others it seems to suffer rather from a general depreciation in common with other parts and tissues, an impairment due apparently to pyrexia or to some diffuse effect of the specific poison; in others, again, from a dissociation from its peripheral complement, from a loss of tone in the "peripheral heart," so that it beats the air in vain. Now, for these different conditions have we any crucial symptoms by which to distinguish them? Have we in a particular case any criterion by which, in an infectious disease for instance, we can discriminate myocardial lesion, myocardial sympathy with a more general and temporary debility, and slackening of the peripheral circula-

tion ? These several factors may overlap each other, cross each other, and supplement each other inconstantly, and in degrees of which appreciation seems very difficult. Nay, we are well aware that in infections—as in diphtheria, scarlet fever, rheumatic fever, pneumonia, etc.—a diagnosis of direct attack upon the myocardium, unless it occupy the conductive tracts, is, perhaps usually, beyond our methods ; if in rheumatic fever dilatation is no infrequent accompaniment of it, in other infections, as in diphtheria, it is at least frequently absent. And in chronic myocardial deteriorations is it too much to say that in the majority of cases of sudden or rapid death the underlying condition is not detected till once for all it is revealed on the post-mortem table ? We have seen that many an ill-looking heart, especially of the fibrous kind, may have worked even against atheromatous stenosis for many a year, and death after all not been directly due to its default. Soft "rotten" hearts are much more apt to "shut up," but how are they manifested ?

During and since my student days attention to cardiac disease has taken various directions, dependent on the various improvements of our means of diagnosis. As a student I witnessed the developments of auditory diagnosis—methods which certainly served to detect many cardiac diseases, such as pericarditis and mitral stenosis, not decisively demonstrable before. Then, by the efforts of Sansom, Ewart, Lees, and many others, especial emphasis was laid on percussion as a means of measuring the dimensions of this chamber or of that—a method by later disciples pushed to extravagant lengths, or used with indiscriminate assurance. The sphygmograph came next, and from it more was expected than it could give ; though twenty years later it was restored to practice by James Mackenzie with largely developed powers ; still, even thus improved, the instrument could tell us only certain things, chiefly facts about rhythm—facts important enough, but, excepting occasionally in respect of *pulsus alternans* or *dissociatus*, throwing little light upon myocardial values. Then pressure gauges were to open out the way for us. Inventors made and improved these instruments, and much was attained in measurement of maximal pressures and even of pulse amplitudes ; but after a while we found that we were as far perhaps as ever from appreciating the

values of cardiac reserve; as much as ever in cardiac disease we were taken in surprise by sudden deaths. Now, again, the X-ray method, and the method of the electrocardiograph, are to place in our hands tests of myocardial dynamics; but hitherto we have had to be content with records which, however interesting otherwise, do not give us the intimate criterions which we seek; for cardiac potential we are still without a gauge.

From these introductory remarks it will be apparent that I do not intend to dwell upon what we call broadly the valvular diseases of the heart. In the valvular diseases we can often, generally perhaps, form some approximate forecast; the conditions are more frankly and audibly mechanical: in these cases the myocardium may count for much, but, generally speaking, it is the static structures rather than the dynamical potentials which take the crooked way. If in a few instances, as in aortic regurgitation, the heart may drop without warning, yet, as a rule, valvular diseases of the heart do not end suddenly; as a rule we may rely on it that a comparatively intact muscle will fight with fairly good reserve energy till defeated by accumulating venous stresses, and by those regional falls in velocity often known by the inept term of "backward pressure." Now of these shortcomings we have for the most part in symptoms and signs no insufficient indications.

From what I have said then it will appear that in the class of cases to be discussed I cannot dwell upon symptoms; for the point is that, even in the gravest cases of myocardial lesion, symptoms may be none; still, there are a few remarks of a critical kind to be made concerning alleged or occasional symptoms. Moreover the problem of sudden death—that is, of absence of symptoms during the main run of a myocardial lesion, even to an extreme period—is not materially altered, if we include certain other cases in which indeed some symptoms do precede death, but do not put in an appearance till the lesion is far advanced. Dyspnea for instance, or falterings in the pulse, or again syncopic phenomena, if they appear at all, may not, and often do not, appear until the malady has entered upon its final stage; so that the issue is removed by but one step from death, and the problem little varied.

But, before considering these admittedly late and almost

irremediable symptoms, let us dwell for a few minutes upon any symptoms or signs from which more timely warning may be obtainable, such as dislocation or readaptation of the chambers; murmurs, unless as superfluous signs of demolition, we may disregard. Fluctuations in the intensity of a murmur depend on so many conditions that, unless other evidences of asystole be present, no reliance can be placed upon such differences, even if referable to any standard. Hypertrophy of the ventricles is so alien to intrinsic degeneration of the myocardium that we may pass it over; but in passing I would remark that, from our present point of view, the alleged intrinsic instability of hypertrophy—say of the left ventricle—apart from the general instability of the diseased individual so affected, seems to me to lack any serious proof. Apart from the causes and conditions of the hypertrophy in the particular case, I know of no evidence that the mere hypertrophy depends upon any encroachment on the normal reserve of the heart, or is taken out of it. So far as evidence yet goes, a soundly hypertrophied myocardium is as good as a normal muscle; a persistently hypertrophied myocardium, as such, does not stand at any intrinsic disadvantage; if the need for it be temporary, the part will recede to its normal dimensions as if no such change had ever taken place. Whatever we may guess, we do not know that it is more easily fatigued, or more liable to degenerative disease; it stands in the disadvantages in which all parts of an embarrassed organ are involved, it is involved in a growing default not its own, and no more.

The heart beat, the impulse against the chest wall, is not a cardinal point, either way. The subcostal impulse is not a simple function of the ventricular contraction; the blood propulsion impulse is not precisely the same contractile movement as the wall shock. It is common knowledge that a well-marked chest impulse may be coincident with a small and bad radial pulse; and conversely.

Dilatation we cannot dismiss so readily (p. 14). It is true that there is nothing very stealthy about a dilatation which is an accommodation for an excessive output or residuum; indeed we need not take alarm at a dilatation suggestive of a loss of tone, if we have reason to believe that it depends on this only, and not upon a textural dissolution. If however it be a result

of myocardial disintegration—as for instance in a heart of fatty infiltration in an obese person, a state which is nearly always associated with an unknown amount, often considerable, of intrinsic degeneration also—then we may well be anxious. Unfortunately, in these persons, with their brawny chests, an accurate delineation is not often practicable. But here we have to halt, or even to return upon our steps; the wall of an enfeebled heart, so the unwary would say, must yield, and yielding signify to the careful observer an increasing dilatation. So it would seem, no doubt; but so it is not—not as a rule. It is a strange thing, but one too generally acknowledged to need arguing here, that frequently a degenerate ventricle does not dilate; indeed it may diminish in bulk. There is no direct or known relation between dilatation and tissue impairment (p. 14). As Lubarsch says, the mere size of the heart goes for little; a small and even somewhat atrophied heart may outbeat a large one. Dr. Mackenzie[1] says, "Dilatation is merely evidence of exhaustion of one function, namely, tonicity," and "many severe cases of heart failure show no signs of dilatation." Heart failure, as he well observes, depends upon many coefficients which need analysis; such as blood quantum, diastolic pressure at moment of systole, friction, vessel tonus, and so forth. Even were this not so, by the close attention recently given to percussion in respect of bath treatment, we have learned that, when controlled by orthodiagraphy, its many fallacies become only too apparent.[2] No experienced physician can doubt, it is true, that percussion, taken with other information, may be of great service as a corroboration, but its direct indications are to be accepted very cautiously. Again, we have no definite means of distributing enlargement, say of the left ventricle, between hypertrophy and dilatation, or between the several chambers, if by other considerations we may guess at it.

Of the absence of dilatation in some such cases, even in many of them, there is no doubt; of the explanation there is much doubt. We may wonder how a decaying muscle can keep its tone; for my part I suspect that dilatation depends upon the

[1] Mackenzie, Jas., *Brit. Med. Journ.*, Oct. 20, 1906.

[2] See Weber u. Allendorf, *Deutsche Arch. f. klin. Med.* Bd. civ., 1912. Vaquez's urinary volumes are, I think, open to the same fallacies.

distribution of blood pressures. In any case, we cannot too carefully remember that there are two modes of dilatation, that of yielding, and that of readjustment which essentially is not morbid. Differences may lie in a contrast between focal lesions and diffuse. In any case then dilatation is no gauge of the histological condition of the myocardium; we have admitted that in some maladies, as in rheumatic fever, an impaired ventricle may be attended with dilatation; while in others, as in diphtheria, no such correlation may be found, usually is not. I am disposed to mistrust too ready a resort to logical explanations by severance of one cardiac quality, such as tone, from another; but the endowment of tonicity may perhaps maintain in position a decayed—even an extremely decayed—ventricle. And if a wider percussion area after exertion be a test full of fallacies we are still without a criterion. We cannot agree with the Leipzig school that dilatation is any exact measure of the quality of the myocardium. On the other hand, a temporary overdistension of the left auricle may deceive us by setting up an arrhythmia resembling for the time the perpetual mode.

If then the delineations of the heart's dimensions which we obtain by percussion, or even by X-rays, give us no constant or trustworthy criterion, what of auscultation? Well, we are but little better off. It was in the discussion at Chelsea which followed the reading of this paper that Dr. Hector Mackenzie spoke of the cases of heart failure in persons who, a short time before, after a searching examination, had been passed as sound for life assurance. Yet the text-books complacently repeat that feebleness of the apex beat, falling off in the tone and quality of the first sound, and prevalence in a larger area of a thin acute second sound, often signify myocardial degeneration. They may; but they are neither obligatory nor conclusive; they depend on transitory causes, and cannot be trusted as warrants. Bäumler repeatedly laid stress upon a weak first sound as thus significant, even though the apex beat were undiminished and quite palpable. The apex beat may be reinforced by recoil from the aorta, but surely a weakness of the first sound is often transient. As to defect of impulse, and defect of sound quality, let any experienced auscultator ask himself how often he has found himself misled by ill-definition of an apex beat, by a poor first

sound and a thin second—severally or even together—in patients who proved in the end to be little or none the worse for the variation. Among scores of such instances I will recall two, both in men approaching threescore and ten. One of them had been reduced in health by business mishaps and by a gall-stone abscess; the other after many years of keen and arduous work was out of heart and anæmic. Neither was obviously emphysematous; yet in both the caridac impulse and area were but feebly appreciable, if at all; the first sound was poor, the second thin and extensive. In both cases consultations were held with "cardiac experts," and in both a diagnosis of cardiac degeneration was pronounced, with little hesitation. The one lived fourteen years afterwards, and died at the age of 83 of an incidental illness; the other ten years later, at an age nearly of fourscore, is still giving his distinguished services to his country. In the first case the heart was examined after death and found free even from atheroma. A fagged or anæmic patient may, without any grave affection of the heart, present for a while the defects of impulse and the quality of sounds which in another might truly signify such a flaw. These equivocal signs then are not criterions. In cases of atherosclerosis, with considerable myocardial deterioration, the impulse usually is not reduced, it may be increased. Patients in whom the sounds are good, or in whom a snappy second may suggest no more than a stiffened aortic limb, a condition of no great relative importance, often die suddenly, and at the necropsy the left ventricle may appear stuffed with callosities, and the muscular largely supplanted by connective fibre. Again, the contrast of small pulse and full apex beat need mean no more than a large area of vasoconstriction, as a soft and full pulse may mean peripheral expansion.

There are other signs which are grave enough, if present, but which are not constants, and so not warrants. One of those is "the paradoxal diminution of ventricular pause (instead of prolongation) after an extrasystole" (Hering, *loc. cit.*). This is not a matter of the vagus. Again Volhard states that in myocardial weakness the prosphygmic interval ("Anspannungszeit") is prolonged. He finds this the rule in pulsus alternans, but I am not at all sure about this rule. The records of Mackenzie

and of Lewis do not seem to show it, and by expert sphygmographers it should be verifiable. If it be true, the rule would be of considerable value in several directions. What, I think, might be fruitfully worked at in chronic heart depreciation, by the auscultatory or Pachon's method, is the curve of diastolic pressures.

To turn from signs to symptoms. Syncopic or vertiginous phenomena often accompany cardiac default; but, whether from dyspepsia, slight labyrinthine disturbance, vasomotor atony, or other obscure cause, we are too familiar with this subjective symptom to rely upon it as crucial in the diagnosis of failing heart. If it be an item amid other signs and symptoms of heart disease, it may indeed enter, and enter gravely, into a diagnosis, as cumulative evidence; but, taken alone, or indeed without strong corroboration, it is very equivocal.

Yet surely by the pulse we ought to be able to make a fair inference concerning the state of the heart. Nay, even here again we are baffled. It has often been asserted that the profounder changes in the myocardium are betrayed by an irregular pulse; but as our experience of cardiac disease has widened, we have learnt that in the rhythms we have no constant or regular criterion of cardiac values. Impairment of certain tracts should, so we think, be surely betrayed by altered rhythms, but we have no criterion even here; we have learnt by autopsy that even the tracts of Tawara may be seriously undermined by disease without derangement of rhythm (p. 50); so likewise may the nervous masses in the coronary sulcus. Lubarsch[1] insists, that in old persons with coronary sclerosis, callus, and fibrosis, fatty degeneration of the *a-v* bundle may prove to have been extensive without the least functional disturbance. On the other hand, in old hearts there are subtle and perilous changes below the microscopic horizon, as revealed by the experiments on the stabilities of old hearts as compared with young ones (p. 475). I think then I shall meet with a general agreement in an opinion explicitly given by von Leyden, Bäumler, Ebstein, and many others, that in the pulse we have no guide to myocardial damage, acute or chronic. Under the cover of normal rates, rhythms, and pressures, decay may pro-

[1] Lubarsch, *Herzpathol. Jahreskurse f. ärtzl. Fortbildung*, January 1911.

gress nearly to the utmost; while positive variations of the pulse may mean little.

Notwithstanding, two variations of the pulse are there, which, when present, are ominous; these are a persistent rise of rate and the pulsus alternans. On pulsus alternans I will not dwell, for Traube, and of late Professor Wenckebach, Dr. Mackenzie, and Dr. Lewis, have urged, a little too positively, that this change—the true as contrasted with the pseudo-alternans—is invariably a herald of heart failure (see refs. p. 58). However, as the sign is no common feature of such default, but a somewhat rare one—one more often absent in failing myocardium than present (Gallivardin found ten cases in twelve months)—it is as a clinical criterion inconstant. Dr. Mackenzie warns us that, if imperceptible to the finger, pulsus alternans may be revealed nevertheless in a sphygmographic tracing. It is to be found in the falling blood pressures of hypertrophy with dilatation of the left ventricle. Prolongation of the pause after an extra-systole—extra-pulse delay—which may be measured on the sphygmogram, is a phenomenon of the same order. The only exception to its significance, as a note of disease, is an occasional appearance of it under the action of some poisons. The researches of H. E. Hering[1] on the causes of pulsus alternans, if not strictly relevant, are so interesting that one is tempted to mention them here. The phenomenon depends, so it appears, on variable refractory periods of the fibres, some fibres being late in response, so that partial asystoles are summed up in rhythmical recurrence. Thus vagus stimulation, by slowing beats, though occasionally reinforcing pulsus alternans, may reduce it, and substitute a continuous hyposystole. Such interferences suggest that between the myocardial fibres there are some obscure differences in value—at any rate in certain hearts; or that the vascular supply may be unequal. The accelerans, on the other hand, as it quickens the rate, may increase the alternation, or by modulation of the refractory discords make it manifest. Thus the cardiac nerves can influence the systolic volume not only directly, but, by modulating the refractory phases, indirectly also.

The other way in which the pulse may suggest decay of the myocardium is by a persistent change of rate, whether of

[1] H. E. Hering, *Zeitschr. f. exp. Path. u. Therap.*, 1908–1909.

acceleration or of retardation; though here again we have not a constant but an inconstant criterion. We shall see (p. 50) that the tract of Tawara may be destroyed, or profoundly marred, without perceptible effect on the pulse; and it is common experience that a normal rate of pulsation is consistent with depravation of the left myocardium at large. Notwithstanding, it has been strongly impressed upon me that a permanent change of rate, either way, is ominous of failing heart. On bradycardia, or again on tachycardia, I need not insist—these conditions are better and better recognised every day; I will insist rather on the less familiar sign of simple acceleration. Let the pulse of a certain man be known as habitually 70, at rest 65-70; and let this pulse rate be found to have become habitually ten more—that is, 80-85, on slight movements rising to 90-100 or over; and let this change show no substantial alteration under prolonged rest, let it fall, that is, under whatsoever rules of tranquillity of mind and body, never below 80-85; then heart failure is in the offing. If, moreover, with this acceleration there be attacks of faintness, or of slight dyspnea or vertigo on moderate effort, the failure of the heart's muscle is at hand; and in elderly persons but little amendment can be promised. Of course, before a final opinion is given, any possible nervous influence, or the influence of coffee, tea, tobacco, which in later life often become less well tolerated, or the passage of an infection, such even as a catarrh, will be reckoned with. Two or three cases of this significance have impressed themselves on my memory: one of them in a patient of Dr. George Johnston, of Ambleside, a country gentleman of advanced years, but for his years of great activity, both of mind and body. In climbing over a stone wall into his own park he was aware of some little faintness, which might well have been a transient affair of little importance. However he was persuaded to send for Dr. Johnston, who found his pulse then at a continuing rate of 100-110. There was little or no evidence of general arteriosclerosis, and arterial pressures were normal. It seemed to the patient and his friends that to keep a vigorous man, with all the appearance of health, permanently at rest, or only to allow him to drive out in his carriage, was a serious decision. I saw him with Dr. Johnston twice at an interval of twelve months; and in spite of the demurs

of his friends, who hinted at hypochondriasis or neurasthenia—neurasthenia does not often come on unaccountably in the later life of persons previously healthy—I could not but support Dr. Johnston's injunctions; for if during complete rest the pulse rate were reduced under 100, or sometimes even to 80, on effort it returned at once to the higher numbers. Gradually, in the course of the next eighteen months, syncopic sensations at stool or under any considerable effort, increased upon him; further and more ordinary symptoms of cardiac failure set in, and he succumbed without any abiding amendment. But here again, as with pulsus alternans, we have no criterion; acceleration of rate in myocardial breakdown is an inconstant phenomenon, and indeed in certain peripheral relaxations may continue for some while without any myocardial disease.

We approach a little nearer to a criterion by calculating the amplitude-frequency product, taken in the several positions of the body, lying and standing; and also after certain measures of effort; in a degenerate heart the pulse, instead of quickening, may be retarded. This test merits a more careful trial than it has received. A fall of maximum pressure may in some cases be favourable, if the minimum fall also; but if with fall of maximum the minimum rises we fear lest the heart be yielding. Martinet (*loc. cit.*) urges that the urine also should be valued at the same time; and, as we have seen again and again in these arguments, unless a very broad basis of comparisons be made, variations in other conditions—in the peripheral circulation for instance—may fog our picture. Core puts it (*loc. cit.*) that when between blood pressure, pulse rate, and clinical symptoms there is no correspondence, the fault lies with the heart; but one of my points is that for a long period clinical symptoms may be equivocal or none; and we have seen, earlier in this work, that the relations between pressures and rates are very variable (Vol. I. p. 43, etc.).

Although the familiar test of recovery of pulse rate after exertion is not without value, we see in convalescents, even from non-infectious illness, that recovery rate depends largely upon peripheral tone and other coefficients of stability. In unaccustomed persons subsidence of effort-acceleration is more protracted than in the accustomed. Ergostat and other effort tests (see *e.g.* Sternberg u. Schmidt, *Münch. med. Wochenschr.*,

Jan. 28, 1913) are of little use; the psychical factor (boredom, or expectant attention) is large, and the adjustments difficult. Janowski, after long and various effort tests, was surprised to find, as I had found, how fallacious they are. (See Vol. II. p. 43.)

Of the many other and more familiar symptoms of heart failure, such as dyspnea, slight œdema, albuminuria, cyanosis of ears and lips, fulness and tenderness of the liver, pulmonary crepitations, arrhythmia perpetua, etc., I need not speak; more or less sudden heart failure is too frequent without any of these features; in any case they usually signify late phases of the malady, speaking only too plainly; but too late. Of the many symptoms to be discounted, I will refer only to arrhythmias pertaining to the extrasystolic kind, which in some cases are, to the inexperienced observer, terrific. One of the worst of these clumsy, bustling hearts I examined three or four years ago with Dr. Davies of Histon, in an elderly gentleman, who, after a little treatment, was safely permitted to go about his business; and now I often see him engaged, apparently without disablement, in public or social occupations. Another case, almost as troublesome, I witnessed about ten years ago in a gentleman, now living, then some 78 years of age; at Homburg he made a good recovery from some "gouty" conditions, and his heart practically recovered its steadiness. Almost as I write these words (1912), he has, on the borders of fourscore and ten, successfully weathered an attack of genuine influenza. In these and such cases of this kind of disorder, even if the heart never wholly recovers a normal rhythm, no harm may come. The symptom group of every such case must, of course, be sifted, for, on the other hand, in pneumonia or diphtheria, or aortic regurgitation, even a mere extrasystole with prolonged pause may be the first presage of heart or peripheral failure. Some recent teaching about the comparative innocence of "extrasystolic arrhythmia" must be taken with salt. Again and again in cases of chronic and apparently stable valvular lesion have I found this phenomenon to be a harbinger of evil; especially in aortic regurgitation. Not at once, not perhaps for many months, or even a year or more, yet sooner rather than later, too often it proves to be a sign of breakdown. If a substantially healthy heart may be, and often is, disturbed in

this way by some extrinsic cause, in a faulty heart a new irritability may be a sign of fatigue. In such cases the intermittency does not vanish, or but for a while; it hangs about and never quite takes its leave. If an intermittent heart be throbbing and uneasy, or even painful, there is the less reason to be afraid of it; harmless extrasystoles may pass imperceptibly, but the intermittences of a failing heart are often also unfelt.

Sphygmomanometrical observations have proved, hitherto, to be of no use in the detection of myocardial disease. That under prolonged high arterial pressures the heart may be defeated is true enough; but this is not a primary myocardial decay. A test of blood velocity, which is probably always reduced, would be more trustworthy, but it is reduced in atherosclerosis, and in persons who live, notwithstanding, to advanced ages.

For the electrocardiograph claims in this direction have been extravagant; in time we may get some definite or even decisive signals from it; but the F(T) wave is at best only the electric expression of the systolic act; it gives the duration of reaction, not the outflow nor the energy. The F(T) wave of a heart proved after death to be depraved may be good enough. Einthoven himself is of more chastened temper; but he is still hoping thus to calculate the weights of the right and left ventricles. And there is another possible opening: in general as age advances, the ordinary height of the F(T) wave diminishes in relation to J(R), and in the dying heart it disappears altogether. Dr. Scales in our laboratories thinks that splintering of the curve suggests senile cardiac decay; but one such case at least I remember in a man who some time later was in good health. Again Eiger [1] states that, in dogs, on chloroform or ether inhalation the F(T) wave flattens out, and even may become negative; cardiac metabolism being profoundly altered. Strubell [2] states that up to the age of 50 this after limb of the curve is well marked, and if with increasing years it becomes gradually less, it does not quite die out. If under the age of 50 it wanes, the muscle is somehow not up to standard; the normal proportions of the healthy adult being

[1] Eiger, *Prager med. Wochenschr.*, 1911, Nos. 23-24; quoted *Arch. des mal. du cœur*, avril 1912.

[2] Strubell, "Kl. d. Electrocardiogram," *Deutsche med. Wochenschr.*, 1912, No. 21. (Doubt has since been thrown on these opinions.)

J : F : A as 100 : 25 : 10. On the A wave age, pressure, and heart size have little or no effect. In young people with moderate pressures the J peak is said to be relatively lower, F relatively higher; in old people the reverse. Thus the ventricular coefficient $\frac{J}{F}$ may wax with age, pressures, and size of heart. But these rules are open at least to much exception; *e.g.* in diphtheritic myocardium the electrograph shows no great variation.[1] Strubell advises, if an estimate of myocardial value is to be obtained, that records of manometer, X-rays and electrograph should be compared, on which comparison he formulates for persistent high pressures four rules, as follows: (*a*) high final wave, with auricular wave unaltered, signifies high pressure with good heart; (*b*) the same with a higher auricular wave suggests a further stage of the condition, with growing stress upon the lesser circulation; (*c*) the same with a waning final and large auricular wave, a failing ventricular muscle; (*d*) the same with a negative F, myocardial failure. By ray method a dilated aorta would often then be seen. Kahn of Prague,[2] by obtaining parallel curves of electrocardiographic and heart sounds, found some interesting relations between heart tones and heart work; such relations, as I have said, so long as merely subjective, are not trustworthy nor recordable, but if traced on paper they may give us useful aid. For the present of course such methods are out of the reach of ordinary clinical work. Vaquez,[3] with whom I have had some conversation, contests Nicolai's opinion that the electric curve can indicate the size, if not the value of the heart's contractions. Professor Mines agrees with Burdon Sanderson that the final wave F(T) is not crucial; it signifies the passing off of the process with a difference of potential in several parcels of the cardiac complex towards diminution of the excited state. It is not likely that it would pass off symmetrically or simultaneously in all regions of the muscle.[4] It is in nature accidental, and can be no sign of failing cardiac value. Indeed it can be varied under experiment. For instance, in a typical case of

[1] Rohmer, F., "Electrokardiogr. u. anat. Untersuch. Diphth'herztod," *Zeitschr. f. exp. Path. u. Ther.* Bd. xi., 1912.

[2] Quoted in various journals.

[3] Vaquez, "Sur la signification de l'électrocardiogram," *Soc. de Biol.*, juillet 1911.

[4] Mines, *Journ. of Physiol.* vol. xxvi. No. 3, June 19, 1913.

pulsus alternans, although the unequal values of the two diastolic pauses were seen on the sphygmogram, the electrocardiogram betrayed no difference in amplitude of contraction ; no variation of ventricular energy was apparent. In this conclusion Mines supports the previous opinions of Hoffmann and of W. E. Hering to the same effect. Hering has shown [1] how far stimulus and reaction capacity are not directly proportionate ; and that electrical stimulus is no complete test. As I have urged on former occasions, this is the curve not of the contraction, but of the quantity of the electrochemical changes which determine a contraction. Chemical stimulus, which is not identical with an electrical stimulus, might, if measurable, tell us more, especially in pathological states ; still it would not indicate reaction capacity. As we have to consider heart nerve tones against heart muscle tone, so Hering, Mönckeberg, Aschoff and others point out a distinction between the contractile power of the heart, and the formation of stimulus ; infirmity or even death might be due not to waning contractile power, but to failure of stimulus. Hering [2] says that stimulus (*Reizbildung*) has also its reserves, its potential.

So far then the polygraph and electrocardiograph have done us invaluable service by revealing laws of centres of stimulation and dissociation, but so far have done little for the estimation of capacities, of intrinsic values. Vaquez warns the electrocardiographer not to exaggerate the value of his records, for in his opinion these are of less value than X-ray observations taken daily.

I have alluded to the method of measuring ventricular contraction before and after measured efforts, a method of which the difficulties, complexities, and possible fallacies, psychical and otherwise, are only too plain. It is indeed premature to say, as some are saying, that even from this variation a criterion of the state of the myocardium can be obtained ; we have for instance to make sure of the inclination of the heart's vertical axis (Waller) ; still a waning of this wave, and yet more a negative oscillation, may prove, under due controls, to have a definite pathological significance, especially if the pauses are protracted. On the other hand, a persistence or

[1] Hering, W. E., *Pflüger's Archiv*, cxliii., 1912.
[2] Hering, W. E., *Deutsche med. Wochenschr.*, Sept. 11, 1913.

recurrence of the juvenile type in adult life may signify a nervous factor.

When fibrillation of the auricle has set in, with arrhythmia perpetua, we have before us no doubt a grave degeneration of one limb of the organ; for the cases in which it occurs, and at the comparatively advanced stage when it occurs, it is ominous enough; though as a signal belated. And it seems still necessary to state clearly that auricular fibrillation is not a cause of cardiac failure but one mode of it. Fibrillation of the ventricle indeed is one of the modes of sudden death; it is hardly consistent with survival.

Two other tests of cardiac values have been suggested; Abram's reflex and Livierato's reflex. By the first we chafe the cardiac area with a towel or india-rubber glove, then, if the heart be active, the percussion area should diminish; if it be weak there is no recession, or but little. By the second we make a number of short, deep, sudden strokes in the middle vertical line, xyphoid to navel; the heart's area should move inversely as the cardiac capacity. Surely these methods are open to much fallacy. Étienne[1] however reports a case of a gouty man with poor irregular pulse, dyspnea, and some pulmonary œdema, in whom, to both these reflexes, the heart responded well; he underwent the Royat cure and did well; and continued well, being able to climb hills, and so on.

I must not tarry to discuss the various devices for testing cardiac reserve proposed by Katzenstein, Graupner and others; suffice it to say that, while some of them are inconvenient, none is effectual or even approximately indicative of the available capacity of the heart under observation. The fallacies of unstable peripheral tone, especially under tests more or less vexatious, are alone enough to confuse the results.

It is said that the rise of venous pressure due to effort is greater in cases of cardiac insufficiency; but what standards have we? Such verifications are very difficult, and, when done, we have no analysis of the many coefficients concerned.

How little we can read cardiac capacity from blood pressure records we have seen abundantly in the chapters on Arteriosclerosis. It may be true, as Sir L. Brunton suggests, that in

[1] Étienne, *Paris méd.* t. 46, 1912.

old people a slackening of arterial pressures may signify a gradually failing heart, if, that is, the patient be watched over a long period, and the readings are consistent. Max Brückner [1] has studied these indications in about 200 cases of diphtheria in the Children's Hospital at Dresden. The results were, so to speak, quite capricious; out of them emerged no trustworthy rules of diagnosis or prognosis. Drops of pressure, perhaps vasomotor, passed off harmlessly, while cardiac failures were not definitely thus foretold. In grave myocardial infection a drop of 50 mm. might occur with the heart failure, but of such defaults there were no premonitions; that is to say, clinical was not preceded by manometric evidence, or not with any regularity. The opinion that in acute infections the failure is often peripheral and vasomotor rather than primarily cardiac, has been gaining ground; as for instance in the late Professor Curschmann's clinic.[2] This alternative, or supplement, attributed by some to poisoning of the adrenal bodies with suspense of their function, must not be forgotten; but it would be a dangerous confidence to take such a view of the majority of these cases. In no circumstances is our lack of any clinical warrant of cardiac capacity, and of cardiac stability, more painfully manifest than in the acute infections. Again and again, in such diseases as diphtheria and scarlet fever, young lives are maimed or cut short by heart failure without more definite warning than the vague forebodings cast by the shadows of like cases gone before. The young man or young woman may, so far as clinical observation can tell us, be going on well, and yet be within a few minutes of eternity. A persistent increase or decrease of pulse rate may hint at the danger; but more often there is no such sign of peril. Now, to drop out in this way, a heart must have been gravely affected for some little while before. Again, after an infection as mild as a "common cold," more commonly after an influenza, the patient has no warning that his convalescence is incomplete until he engages in some effort, such as a game of football, and then finds himself invalided for many a month to come. Without any clinical manifestation his heart had become the seat of occult disease. Here again our only guide is crude experience; we

[1] Brückner, M., *Deutsche med. Wochenschr.*, 1909, No. 14.

[2] See also Pässler and Rolly, *Münch. med. Wochenschr.*, Oct. 1902, p. 1737.

have no criterion. Tanaka [1] declares how remarkable it is that a "heart often functions well up to the moment of sudden death," yet after death the myocardium reveals disease both extensive and profound. The ventricle may or may not be dilated. It is a little out of my subject, but I am tempted here to say how marvellously the myocardium recovers after infections in young people. I remember a paper read last year (1912) by Heffter, at the Münster Meeting of the Deutsche Naturforscher u. Ärtze, in which a very competent histological report was given of the heart of a boy who, while recovering from diphtheritic heart, died of cerebral embolism. The regenerative process was very evident, and included a new development of muscular fibres. The paper seemed to me to be one of much interest and importance, for the author followed up the report with more evidence of the same kind.

Thus baldly I have tried, from the point of view of the physician, to show how indefinite, inconstant, and obscure are the symptoms and signs of myocardial insufficiency; let us see then what the pathologist has to say—if with his scalpel and probe he can offer us more definite measures of cardiac capacity. We shall say to him, surely you ought to discover why the patient died? why his heart, or the other man's heart, broke down without warning? or in any case what was the critical point at which its resources could no more avail? But too often in his turn the pathologist gives the same uncertain sound. In this heart he found too little to account for death, in that too much—the patient had no business to have lived so long. On grounds as insecure as those of the physician, he finds one heart which, with comparatively little or even no evidence of decay (Cabot), surrendered at the first summons, or became suddenly insufficient; and, conversely, another which by the very extent and chronicity of decay testified to the long period during which it had managed to wage an unequal fight with silent tenacity. From the side of morbid anatomy then the want of parallel, of apparent correlation, between the physician and the pathologist lacks nothing of its emphasis.

But, at the outset of this side of the matter, I would with

[1] Tanaka (of the Göttingen Pathological Laboratory), *Virchow's Archiv*, vol. ccvii., 1912.

all due deference suggest that in many a report to the physician from the post-mortem chamber, the histological changes described are consistent with, or even declare, rather the effects of the decomposition of death than of disease during life. I have had to discard many a note recording blurred striation, nuclear alterations, "hyaline" change, etc., when recorded without any note of date after death, or of the state of the atmosphere. Indeed so common is the tendency in the viscera to a focal distribution of their lesions that one may wisely, I will not say reject, but regard with suspicion, reports of diffuse and uniform myocardial disintegration, as possibly due to mere post-mortem decomposition.

Of the kind of detriment called "fatty degeneration" opinions of late have undergone some fluctuation; the term, for a while discarded, seems to be returning into use, though with some difference of interpretation (p. 58). The distinction between fatty degeneration and fatty infiltration, though the extremes are rare, still holds its ground, and between them lies every grade of transition. If in the obese heart much of the fat is to be traced to an interfibrous penetration, yet on the other hand in very few such hearts is this penetration unaccompanied with some deterioration of the muscular fibres themselves. And if in "fatty degeneration" the droplets of fat may be but particles of unassimilated nutriment rather than a disintegration of muscle cell, or a metamorphosis of albuminous structures into fat, and if indeed we cannot say that simple fatty accumulation is a myocardial regression, yet even delay of absorption and assimilation seems to signify some alteration of chemical change, if in rate only; and if so to signify some modification of muscular energy. Aschoff however goes so far as to suggest that this fatty manifestation may signify an excess of physiological activity. It is certain at any rate that, in the midst of it, the fibres and their striations may still be well defined (Aschoff, Lubarsch, and many others). In old hearts with coronary sclerosis Kent's bundle itself may be thoroughly "fatty," yet with no apparent conductive decay. Fatty content must be contrasted with fatty decay; it may be due to a passing torpor of function, or to slow arterial and lymphatic circulation; or occasionally to the arrest of death. The vacuoles we

read of are probably artefacts. The nuclear reactions should of course be tested. In diphtheria, beside the fatty deposits, the muscle fibres also are obviously diseased. In some toxic cases it would seem that the "fatty" state may be established in twenty-four to thirty-six hours. However, what I have to emphasise is the agreement that even well-marked fatty deposits, severe and diffused, may appear without any clinical evidence of heart failure; so that in this context pathology can provide us with no criterion. That, as I have recently seen stated, the "fatty heart" is to be known by dilatation or by irregularity of rhythm cannot be maintained (p. 34); these coincidences are quite inconstant.

In rheumatic myocarditis (Romberg, Cowan, Carey Coombs) sudden heart failure is less common; usually it is so combined with dilatation and valvular disorders that clinical symptoms are not wanting. Interesting as the changes are from a histological point of view, I must not in this context dwell longer upon them. Nor do the distinctions, definite or transitional, demonstrated by Dr. Carey Coombs and others, between acute interstitial and parenchymatous myocarditis much concern us here. We will pass on to chronic myocarditis.

In so-called "chronic myocarditis" again, probably not strictly an inflammation but partaking rather of an atrophic or degenerative nature, we find no closer correlation between the clinical and the pathological series. Cabot, in a very searching report[1]—one in which he compared clinical and pathological notes taken independently—says that he has quite given up attempting to diagnose "chronic myocarditis"; "in it diagnosis is a mere matter of luck. It has no clinical symptoms, no characteristic physical signs." So this point needs no further pathological discussion.

But from the clinical point of view I must return for a moment to fibrosis of the myocardium, a change which an eminent student of heart disease but a short time ago assured me was of great clinical importance. To this I demurred, for, as I have said (pp. 11-18), this fibrous invasion may be wholly without clinical correlations. Many years' experience has convinced me of the truth which Cabot again (*loc. cit.*) has

[1] Cabot, *Massachusetts Gen. Hosp. Reports*, October 1911.

lately emphasised; namely, that "not infrequently gross fibrosis of the myocardium is found in hearts which had been perfectly efficient during life." Dr. Hale White [1] also says of a fibroid heart that it is practically undetectable. This paradox is true of both the forms of cardiac fibrosis of which I have spoken—of the diffuse form described by Dehio [2] in 1899, and the focal form, in which the wall of the heart, especially of the left ventricle, is beset with callosities; though this, if extensive, is by far the graver lesion. These callous nodules or lumps were at one time attributed only to local ischæmia due to a closure of a coronary branch, by infarct or by sclerosis. Histologically however they do not essentially differ from "myocarditic" effects of other origin; moreover, recent research has demonstrated many a callus towards which its particular coronary twig was open (pp. 21-23).[3] But these nodules, often old foci of toxic myocarditis, are not always to be found by simply slicing the ventricular wall with a knife; sections must be made and stained. Conversely, Kanthack and I, in investigating the effects of coronary disease on the heart, demonstrated repeatedly that these arteries might both be very gradually blocked by atherosclerosis, and yet the myocardium keep normal enough for the age of the patient. Clinically speaking, with such hearts patients may, as Dr. Hale White says, die suddenly; but we have no criterion of the change, nor can the pathologist tell us how or when it causes death, if indeed it does directly cause it; which is doubtful.

Diffuse or dystrophic myocardial fibrosis is a different condition. Dehio attributed it to venous stagnation, comparing such hearts with "cardiac kidneys" and the like; and it is true that dilated ventricles are often fibroid. Still, I have examined many hearts presenting this fibrosis in which evidence of venous congestion was not obvious. However, I must not leave signs for points of pathology which from that point of view I have dealt with already (p. 9 *et seq.*); in respect of symptoms suffice it to say that cardiac fibrosis, so far from being a cause of

[1] Hale White, *Clin. Journ.*, November 29, 1911.

[2] Dehio, *Deutsche Arch. f. klin. Med.* vol. lxii.

[3] Ebstein, "Schwielenbildung im Herzen," *Zeitschr. f. klin. Med.* Bd. ii. S. 97; Fahr, *Virchow's Archiv*, February 1909 (and Bäumler, Gierke, and Ziegler agree). For presence of fibrosis in hearts which probably were efficient see (p. 18) Gennari, *Arch. per sci. med.*, December 1905.

cardiac failure, seems not infrequently, as in hypertrophy with dilatation, to have some conservative value. We have seen that connective fibre is a less resilient tissue, and its elastic limit narrower, so that once overstretched it cannot well recover ; but it is tougher. Syphilitic fibrosis is, of course, another matter ; any effect this variety may have on the cardiac function depends on its site. "Cardiosclerosis" then, whatever its propriety as a name for this condition,—one however to which it has not been definitely attached: I have said that we never succeeded in ascertaining precisely from Huchard, or from his disciples, what they meant by the name,—has in this sense no clinical connotation. It was adopted for logical symmetry rather than to express the complexities and elusiveness of nature.

Let us now pass on from the histological nature to the seats of lesion. Is it less the nature than the position of lesion or decay which determines the mortality ? Here again I fear the answer of the pathologist is still equivocal, though it is true that closer methods of observation may prove more enlightening. The lesion, or the critical lesion, may be overlooked ; for instance, Falconer and Duncan,[1] beside valvular lesions in a certain heart, found diffuse lesions in the right auricle (less in the left), while, save for a slight perivascular lymphocytosis, the ventricular muscle was normal.

In respect of site our thoughts turn promptly to the tract of Tawara, and perhaps to Wenckebach's area about the superior vena cava, for we must not be content to say that lesions in this area set up disorders of rhythm only. We readily suspect, in such a concert as the function of the circulation, that rhythmical discord may abut upon and issue in defective work. At first sight it seems that even if scattered focal lesions—such as those described by Carey Coombs, Poynton, and others—come short of gravely disturbing conduction, yet such a check must follow a more diffuse and general invasion of these structures ; for ventricular automaty, detached from the cardiac system, is certainly less effectual. Many cases are published in which cardiac function persisted normally in spite of what appeared to be (as in Dr. Raw's sarcoma case and many others) complete destruction of the conductive tracts (p. 36). But to say in a

[1] Falconer and Duncan, *Heart*, vol. iii. Part II. para. Tachycardia.

particular case that the destruction was complete is to beg the question; only a jury of experts could come to this decision (see note 3). Syphilis, by its proclivity for the septum, especially menaces Tawara's tract. Now some observers are of opinion that this tract, whether by its looser texture or by its peculiar blood supply by a branch from the right coronary artery, has in its disease some independence, be it more or less, of the rest of the heart.[1] Spots of hæmorrhagic and leukæmic exudation, aortic rheumatic foci, and the like are found not infrequently in this tract, especially in the left limb of it, when absent in other quarters of the heart. Tanaka insists, as I have done, on the curious maintenances of heart function and of medical confidence in not a few cases in which the myocardium and conductive tract proved to be in extremes of fatty degeneration, until in a moment death brought all to an end.[2] This, he says, as Dr. Hugh Anderson had illustrated in the cat (Vol. II. p. 475), is especially characteristic of old as contrasted with young hearts.

But we must not, I think, make too much of this alleged independence; decay proceeds as often from the myocardium to the bundle and knot, and again, as Dr. Coombs has shown, both tracts and myocardium, if unequally affected, are often attacked simultaneously. Saigo[3] examined 100 hearts from this point of view, and concluded that rheumatic nodules do often arise in the bundle; that regressive processes also—such as fatty vacuoles—come early in it; but that in brown atrophy and fibrotic hypertrophy the bundle may be involved, or may be free, or nearly free. Lewis, Coombs, and others have shown by careful methods that transient ventricular lag may be revealed in such infections as rheumatic fever, diphtheria, or influenza; yet they will probably agree that such clinical phenomena are rare, and when present indicate the seat rather than the degree of the lesions. A block of slight degree may coexist with widespread myocardial injury; and conversely. Frequent as are dots and foci of toxic lesion in and around the conductive strands, alterations in the auriculo-ventricular sequence are surely exceptional (Poynton, Coombs). And in

[1] See Tanaka; *loc. cit.*

[2] These anomalies Prof. Kent has since explained, *B. M. J.*, July 19, 1914.

[3] Saigo, *Ziegler's Beitr.* Bd. xliv.

young hearts, under proper treatment, happily they are usually transient, and are completely repaired with the formation of tiny scars, and even some redintegration of nobler tissue (p. 46). In old hearts of course the mischief may be irremediable, but on this well-worn subject I need not dwell. A patient of Dr. Canney of Cambridge, aged about 65, whom I also saw repeatedly, seemed to recover from a grave attack of scarlatinal myocarditis. For some months his state was perilous, but he got actively about again, and after six months' absence from work and change of scene, he returned to his usual exercise, bicycling quietly up and down hill, and the like. Some months thus passed with no sign of ailment or of morbid cardiac action ; but one evening after complaining for an hour or two of feeling very cold, he went out to certain duties, and in the midst of them turned faint, rose to leave the room, and died in a few minutes.

Lubarsch [1] on this complicated problem investigated 500 hearts ; in many he found changes, such as acute rheumatic nodules, predominant in and about the tract, especially the left limb of it. Brown atrophy he found to occur in both myocardium and tract. His observations may be summed up thus : in moderate focal fatty myocardium the limb of Tawara's tract may be quite free ; or in extreme and widespread focal fatty myocardium the *a-v* bundle may be very slightly affected ; or fatty change may be quite isolated in it, even the papillary muscles being free (but this is rare) ; or fatty change may be marked and extreme in the *a-v* tract, with very slight myocardial affection, say slightly in the papillary muscles only ; or fatty change, moderate or much, may be practically alike and equal in both myocardium and bundle ; finally, Lubarsch thinks these variations are dependent upon vascular conditions, such as local retardations of blood velocity.

In Tanaka's cases of diphtheria affection of the bundle was five times more, three times less than the rest of the musculature, and four times the incidence was equal. " Only once was an almost isolated bundle-affection found." In the bundle the changes are more diffuse. " But," he concludes, " a sudden failure of the heart can also come about without essential lesion

[1] Lubarsch, *Jahresb. ärzt. Fortb.*, 1911. See also *Amer. Journ. Med. Sci.*, 1912.

of the bundle; occasionally indeed arrhythmia appears without any marked degeneration of the bundle."

In one of Lubarsch's cases, in which dissociation was complete, while in the myocardium were found after death large focal callosities, in the bundle were only small ones, with some fresher spots of exudation and minute hæmorrhagic points; none extensive enough to rupture continuity. On the other hand, many cases are published (by Reinecke and others [1]) in which, under what seemed distinctly to be complete demolition of the bundle, there was during life no dissociation. In one case [2] the whole bundle was calcified; in others in which the tract seemed broken up dissociation was often incomplete; sometimes there was only slight retardation. Dr. Coombs' researches again make it probable that acute rheumatic or septic toxins may far invade the tract without any characteristic symptoms during life. Conversely, cases of heart-block occur in which expert pathologists discover no disease in the bundle.[3] If one limb of it is affected there may be incomplete dissociation. In a few cases tachycardia has been observed; I have seen two such instances. I will consider presently how far intrinsic and extrinsic causes (bundle, vagus, bulb) must co-operate; or again how far chemical perversions may be concerned in arrests of the heart.

I make no excuse for dwelling at this length on the pathological relations of the bundle and the myocardium, for in them we may find some explanation of the obscurity of the causes of continued cardiac function on the one hand and of sudden cessation of it on the other. In some bundle cases, probably in most, the proper symptoms of dissociation are present, but in some they are absent; and as yet we have no histological elucidation of this inconstancy (see however p. 51, note 2).

Attempts have been made to test myocardial values by drugs, but with little that is promising. A drug may affect the heart muscle directly or through its nervous machinery, motor or afferent; it does not seem probable that we shall make much out of this kind of test. We cannot establish controls.

I have alluded more than once to the part of the nerves

[1] See *Deutsche Arch. f. klin. Med.*, 1908.

[2] Nagayo-Aschoff, "Adams-Stokes Sympt.-komplex," *Zeitschr. f. klin. Med.* Bd. lxvii.

[3] *E.g.* Fahr, *Virchow's Archiv*, 1907, vol. 188.

in cardiac function, and so in the clinical surprises which we are discussing; but the nervous coefficients in these functions are very dark. Is it by means of nervous influence that a heart which is the seat of advanced disease may be carried along so far and so efficiently as again and again to deceive the clinical observer outright? We know that the heart is thickly beset with nervous ganglia and plexuses, that these are motor in function, and that much, if not all, of the harmony of the circulation must depend, and intimately depend, on their integrity; but our new interest in Kent's and Tawara's tracts has diverted research from these nervous governors. We may destroy aortic cusps without immediate heart failure, we may poison the myocardium into extreme fatty change without immediate heart failure; but if with the soundest myocardium we cut the vagi, or even cut one only, the organ may promptly stop—at any rate in dogs and cats.[1] Kronecker's experimental determination of a nervous co-ordinating centre in the upper third of the interventricular septum is well known; a needle puncture here (in the dog) produces fatal delirium cordis, a result not to be obtained by puncture anywhere else. The morbid histology of the nervous cells and afferent and other fibres of the heart is little understood; even their normal anatomy is vague, and the pathological records are dubious. Our methods may be inadequate, but hitherto the allegations of morbid changes in the nerves do not amount to proof. Vagus alterations have been reported, but on scanty evidence; yet if the nervous strands and centres deteriorate, and we cannot suppose them to be immune to the lot of all organised structures, their irritation or decay may account for a waning cardiac reserve, and for unexpected heart failure, even in cases in which muscular degeneration may be inconsiderable. In degenerate hearts Romberg, Krehl, Rosenbach, all turn for explanation of the paradox of endurance, and of deferred crisis, not exclusively to the "immanent properties" of the organ, but also to its nervous endowments, and especially to its ganglionic integrity. Whatsoever our opinions on the rather academic controversy between the neurogenists and the myogenists, we shall not forget that the

[1] Among many such records see, for example, Balint, *Deutsche med. Wochenschr.*, January 6 and 13, 1898, and Vol. II. p. 475 of this book.

nervous system of an organ, such as the heart, is not only the conductor of its numerous and harmonious concert, but also an instrument of its development. To Gaskell and Professor Langley we are chiefly indebted for the knowledge we possess of the sympathetic system, and Langley tells us that although the ganglionic cells of the heart comport themselves differently from sympathetic cells, yet that if deprived of nutriment these ganglia die rapidly; as for example in animals bled to death. They have, it is true, a greater resistance to deprivations of oxygen than has the central nervous system, yet oxygen may be more important to them than to nervo-muscular tissue.

And, beside the intracardial nervous structures, we have mentioned the extracardial, the vagus and the bulb; and, above these, the psychical influences which on the heart are supposed to have a peculiar potency. One would guess that a heart failure attributable to these influences might be due to a cortical disturbance propagated through the vagus (Vol. I. p. 70).

Furthermore, the vasomotor system has reinforcements and defaults of which we have little comprehension. Many a syncope, or death, attributed to the heart is due to a failure of the "peripheral heart," to a relaxation in large vasomotor areas. For such cases a clinical test for adrenin is wanted. The "weak heart" of women is often thus to be explained; their peripheral vessels are labile and slack. Here again one is tempted to dally with the hypothesis suggested by Henle, and ingeniously advocated by Hasebroek and Grützman [1] (see Vol. I. p. 51), that the peripheral heart—the muscular arterial net—acts, not only as a break on the central propeller, but also as forwarding machinery, a subsidiary means of propulsion.

Another side of vascular dynamics is not fully seen, namely, the reciprocation of the several areas; yet these mutual tides are ubiquitous and incessant. I have often observed that if the warm hand be placed between the thighs, and the pulsations in it felt for a few minutes, oscillations of vascular volumes, independent of respiration, will become clearly perceptible. And Müller [2] and Weber [3] have shown that areas are very

[1] Grützman, *Deutsche Arch. f. klin. Med.*, 1906.

[2] Müller and Weber, *Arch. f. Anat. u. Physiol.*, 1905.

[3] Weber, *ibid.*, 1906. References to well-known English writers are not necessary.

independent, often indeed in conflict, and that such pressure interferences have their echo in the heart; if in an area dilated for work velocity is not to fall, there must be an equivalent and harmonious constriction elsewhere, or the heart will have to make up the difference (p. 30). The mere movement of a limb determines at once an afflux of blood thither.

Furthermore, we must not forget the chances of sudden death by fat embolism, nerve toxins, pulmonary embolisms, and so forth; or possibly, as suggested by Dr. Hawkins-Ambler, by increases of the specific gravity of the red corpuscles. There are probably many subtle agents, of which we have little notion, which enter, alone or together, into these nice poises of life (see chap. Viscosity, Vol. I. p. 112); and likewise alterations in molecular constitution invisible even to the "ultramicroscope." An old rubber band may to the eye show no change, but on use its molecular change will become apparent; it will die suddenly. The heart of Dr. Hugh Anderson's elderly cat could not withstand vagus inhibitions to which the young cat reacted indeed plainly, but which it withstood with security (p. 475). Beyond our reckonings, within the visible features, there is a world—a world of surface tensions, osmoses, reaction potentials, viscosities, differential pressures, and the like. If we apply cold to the sinus area, we retard the rate of the whole heart; if heat, we accelerate it, as in fever; yet to the eye there is no tissue change; and to other parts of the heart such applications of heat or cold are relatively indifferent.

Still, if sound and not too old, the heart, as Sir James Goodhart declares, will stand almost anything; no combination of stresses seems too much for it. The distinctions urged by Merklen, Hoffmann, Rosenbach and others between anatomical and functional cardiac integrity are pushed too far; there can be no function without correlative molecular motions, and if the function continues irregular, well, the corresponding molecules have not returned to their normal pattern; there must be some dislocation, and this may be permanent—a "new set." I am told that if by repeated blows a piece of steel be fatigued, although to the microscope no molecular alteration may be visible, yet nevertheless, by a change in its electric conductivity, a permanent molecular dislocation may be detected.

Perhaps some day we also may be enabled by the test of electric conductivity to gauge an old, a fatigued, or a poisoned heart. If the texture of a heart has got a permanently new set, whether the intimate alterations be visible or invisible, it is no longer sound.

Another curious point about cardiac reserve, or what we think to be such, is that it is trainable. A man in training seems to have many times the reserve, the cardiac reserve, of one out of training. In athletic training the heart probably grows larger with its skeletal kindred, and therewith no doubt the tone of its motor ganglia and of the peripheral vascular system; and the capacity of the lungs is developed also. In Cambridge an old boatman used to test an oarsman for staleness by drawing a finger down his cheek and noting the persistence of the ruddy line. However athletic training is not a mere matter of heart and lung endurance; the acquired economies of the skeletal muscular groups count for much. We cannot yet analyse all these manifold factors. In some elderly persons the skeletal muscles seem to have kept their vigour almost too well; the heart was not able quite to keep up with them.

Then there is the world of chemical reactions in the heart; the ionic motions and substitutional interchanges of which we know little: but, from the known behaviour of salt perfusions, they must be of the most intimate kind. Such researches as those of Dr. Walter Fletcher and Mr. Barcroft are bringing some light into this subject. Dr. Gaskell showed years ago that to perfuse the heart with even a very weak solution of lactic acid is to stop it in diastole; a reaction due to mere acidity, to the positive ion H. Stronger solutions drive it into rigor. And Dr. Fletcher has shown how by a previous application of oxygen these effects can be prevented.[1] The mammalian heart lying after death in an atmosphere of oxygen, "so long as the normal architecture of the muscle is maintained," does not become acid, or but slightly so; and after perfusion with oxygenised Ringer's solution, and even some hours' pulsation, no trace of lactic acid appears. In the fatigued but normal heart on the access of oxygen the lactic acid promptly disappears. Dr. Fletcher has shown me how many occult flaws there

[1] See Fletcher and Hopkins, *Journ. of Physiol.*, March 27, 1907.

may possibly be in the balance of colloidal mechanisms in the cardiac muscle; and more than this—that the heart may have abundant fatty deposit in it, and yet the muscle cell on the motor side be, as we have said (p. 47), intrinsically intact, and prove no more vulnerable than one histologically normal. This he illustrated by the figure of an engine clogged with dirt and grease, which, notwithstanding, may do better work than another engine bright and clean, but with tight bearings or an unseen crack in its cylinder. I repeat, as Welch and others have shown, that moderate degrees of grey and fatty change are compatible with fair heart work. But when, beyond perverse metabolisms, we pass into the region of toxic influences we recognise again how near the edge our gyroscope may revolve.

Finally, we know that the heart's balance depends on that of many other parts also; the gaseous exchanges are not in the myocardium only, not even in the lungs only—they are burning in every muscular area, in every viscus. Thus, even if the hæmoglobin be constant in quality and quantity, if velocity begins to fall we are in a vicious circle; velocity—which means oxygen exchange—slows down in every organ, especially in those which, beside the lungs, have a double capillary system, such as the liver and the kidneys; in these areas we very soon find evidences of stasis. And retardation in the lymph system also is not to be forgotten. Such defaults as these may for whiles miss or neutralise each other, but when at an unhappy moment a summation of alternating conditions comes about—capillary retardations in the viscera, tissue calls, lymph pressures, vagus and vasomotor waves, psychic thrills—in such cases as these, cases not without their forebodings, we may not be at a loss to comprehend the suddenness of death. But the riddle, which I have done so little to read, is the frequent suddenness of death in one who, scarcely having known illness, expires under no extraordinary effort; or in the peace of his own bed or chair passes silently away. The reading of this riddle is not yet.

P.S. to p. 37. Further, on pulsus alternans and extrasystole, and on the forecast, see two papers by V. Hoeslin and O. Roth, *Deutsche Arch. f. klin. Med.* 114, 1914; also Gravier's treatise, 1914.

CHAPTER IX

DIAGNOSIS AND PROGNOSIS OF ARTERIOSCLEROSIS

Diagnosis.—The bent of mankind to deal with things not as facts and experiences, but in an abstract, logical, and even fanciful fashion, is very curious; passing over a transitory feeling for reality in ancient Greek lands, and again in the thirteenth century, it was not until the sixteenth that any systematic and persistent attempt was made to observe things as they are. Curiously ingenious as have been the logical and speculative categories of the pulse in ancient and even in comparatively modern times, many of them indeed being based on more or less accurate clinical perceptions, yet our remoter forefathers took little heed of what seems to us so plain, the various thicknesses and resistances of the arterial tunics (see Vol. I. p. 3). An appreciation of such changes, coinciding more or less with the experiments of Hales, Chauveau, and their disciples on arterial pressures, somewhat suddenly created a new field of diagnosis. But in the preceding pages I have implied, or even directly indicated, so much in comparison of symptoms that I need make no long repetition of such matter. Concerning diagnosis then I will confine myself to a few brief and desultory remarks on certain points of emphasis or of application.

The first broad distinction to be made is between *the hyperpietic, the toxic and the senile or decrescent sclerosis*. The different issues of these three kinds I hope I have made clear. The toxic modes are often so closely associated with an infectious or poisonous cause as to carry their diagnosis with them; as in the cases of typhoid fever, syphilis, or lead. In these cases, as a rule, the blood pressures do not rise. As regards the toxic varieties, I may urge the reader once more not to forget the wide prevalence

of syphilis, and the value in any doubtful case of the Wassermann test. He will not forget, for instance, that a virgin may unhappily become infected; by fondling a syphilitic baby for example, or by some other chance, as in medical or nursing duty. We must not take any person to be outside the possibility of syphilis. Many persons so infected are wholly ignorant of it; many indeed who have incurred the disease by their own fault are unaware of their punishment, and from some of these the truth must be concealed. And as Dr. Mitchell Bruce says,[1] even in a case where a past syphilitic infection is known, we may have to unravel a complexity with metabolic or other perversions of function.

The so-called "neurotic" or "functional" cases of high pressure are only high systolic pressure; the diastolic is unchanged, or lowered. The level fluctuates widely.

Gibson used to demur that my divisions of hyperpietic and decrescent disease were wanting in sharp boundary lines. Well, where in animated nature are such lines to be found? Between these two chief classes indeed the divisions are rather sharper than is common in nosological classification. But it may not always be easy to discriminate between hyperpietic and decrescent arteriosclerosis in the cases where these two processes co-operate. However, the *mixed or intermediate cases* are rare: the following seemed to be a case of mixed senile and hyperpietic disease:

Rev. C., æt. 68. Patient of Dr. Ealand of Farnham. For a year or more had found his mental powers failing; became depressed in a morning, better towards evening. Cannot pursue his thought; *e.g.* he can preach an old sermon well enough, but cannot write a new one. Also cannot walk so far; fatigues in a mile or so. Wakes too early (4-5 A.M.). Andrew Clark had told him ten years ago that he suffered from suppressed gout. Spare and rather pallid man. Gait quite normal to all tests. I saw this gentleman several times, and, to sum up the conclusion in a few words,—the blood pressures ranged, both systolic and diastolic, above normal, say to 180 as highest systolic. But his heart was obviously yielding; it was widely dilated to the left, and murmurs (systolic) were audible at both orifices. The second aortic sound was ringing, over a large area. The accessible arteries were large and leathery, contained

[1] Bruce, M., *Second Luml. Lect.*, July 15, 1911.

calcified dots, and, to touch, suggested Broadbent's "virtual tension." At first sight I regarded this case as simply decrescent; but on consideration I felt sure that it was in the first instance one of hyperpiesis, on which the decrescent series had engrafted itself: or the two processes may have been practically concurrent. How far the heart failure was dependent on coronary disease, how far on the stresses of high pressures, probably never extreme, could not be decided. The large left ventricle pointed at least to no inconsiderable coefficient of blood pressure (see also Episodic Hyperpiesis, Vol. I. p. 453).

In *diagnosis* of **Hyperpiesia** it seems paradoxical to say that a disease of high blood pressure and hypertrophied heart is often mistaken for neurasthenia; yet such is not rarely the case. As it happens that my attention has been given to both these maladies my opportunities of observing this confusion have been many. And the dilemma is by no means always one of confusion of terms. To distinguish in a particular case between *hyperpiesia in its earlier stages and neurasthenia* may be a matter of some difficulty; in an impressionable subject it may be almost impossible to secure a pressure record free from the fallacy of nervous exaltation, and to discriminate in their early stages between cardiac enlargement and irritable heart, especially in women with large breasts. Moreover in such persons the aortic second sound, on account of some laxity and larger diameter of the vessel, is frequently accentuated. Dr. Theodore Janeway, in his well-known work, rightly criticises "the reported high readings in neurasthenia"; and points out that these may on the one hand be due to transient nervous impressions, or on the other to vascular or renal disease. In neurasthenia, he says quite truly, the blood pressures, if they can be taken in tranquillity, run rather under than over the normal values. One criterion is that if in neurotic persons the systolic pressures are apt to run high, especially under examination, the diastolic are unchanged. Dr. Eric Macnamara,[1] in an article chiefly devoted to therapeutical means, comes to similar conclusions. His pressure records in neurasthenia were often minus, but sometimes plus; and were very variously affected by treatment (chiefly electric). In reading his paper I could

[1] Macnamara, E., *Lancet*, July 18, 1908.

not avoid suspecting that some of the cases regarded as neurasthenic were in fact early stages of hyperpiesia; in these high frequency currents are often followed by a reduc- of the arterial pressure (see Vol. I. p. 441). By precise ortho- diascopy enlargement of the heart may be ascertainable before it becomes manifest by ordinary physical signs. We must remember also that hyperpietics are themselves prone to nervous exaggerations of systolic blood pressure, so that for sure diagnosis the records must be patiently verified. Von Jaksch also reminds us that these patients are often "nervous"; they may apprehend ill-health, or interference with their work in life; in such conditions after the patient has become more used to these investigations the excessive pressures slowly fall, and under treatment the instability may prove to have been temporary. Notwithstanding, I do not feel sure that von Jaksch fully discerned the differences between hyperpiesia and neurasthenia; for in another place he speaks of "neurasthenia" as one of the first symptoms of arteriosclerosis; thus using the term not in its technical sense, but too literally as mere debility or weariness. In this sense "neurasthenia" becomes one of the first, or last, symptoms of any malady whatsoever—a futile proposition. Dr. Stengel scarcely avoids ambiguity in this respect. Occasionally then one sees false diagnosis of hyperpiesia in cases which are really but neurasthenia with unsteady cardio-vascular conditions and high systolic pressures. When a well-known foreign physician writes that neurasthenia is a consequence of sclerosis of the cerebral arteries, he throws all nomenclature on the scrap heap. Mere local vasomotor tides, such as cold hands and feet, flushes of the face and the like, are of course independent of the general arterial pressure. Paræsthesias of the legs often occur in both maladies. As an instance of the dilemma we are discussing, I may allude to a case which I saw some years ago with Mr. Winwood Smith of Torquay:

Mr. ——, about middle life, a gifted man of a very highly strung temperament, and of a very "nervous" family, had been "neurotic and dyspeptic all his life." So far the diagnosis lay in little obscurity. But recently Mr. Winwood Smith had found his arterial pressures ruling high—170-180; I verified these readings,

taking such imperfect precautions against artificial exaltation as in a consultation were practicable. And these observations were reinforced by an unquestionable increase of the heart to the left. The aortic second sound was ringing; the rhythm was rather laboured, with an occasional extrasystole, and a disposition to reduplication of the first sound. To the touch, the arteries were in fair condition. Under diet and other remedies these symptoms were ameliorated, and indeed disappeared. A few years later this gentleman, previously unknown to me, became a friend and neighbour, so that I had many opportunities of determining his condition; and although he had now and then to seek for medical advice, the hyperpietic condition did not return.

Anæmia is not, as a rule, a feature of hyperpiesis; there are, as I have said, sallow hyperpietics, but speaking generally anæmia would rather suggest renal disease (see Vol. I. p. 452).

We shall not forget the possible influence of variations in *glandular activity* in some cases. The records of blood pressures in Graves' disease are inconsistent; some observers postulate in this disease a definite and persistent rise of pressure, others deny it.[1] That in particular cases of Graves' disease the systolic blood pressures are often exalted is certain, but this is rather a frequent incident of the disease than a character of it. The diastolic and mean pressures are usually moderate, or even low.

If neurasthenia may be confused with hyperpiesia, or conversely, and if again even worse diseases, such as General Paralysis of the Insane, diffuse cerebral syphilis, or incipient dementia, may at their beginning be confounded with neurasthenia, we perceive that in particular cases some nicety may be required in deciding on the diagnosis. It would be out of place here to enter upon the large field into which these distinctions would carry us, and happily I can refer the reader to a far better appreciation of these complex factors in Dr. Mott's article on the subject.[2] In some of these cases the Wassermann test may be required, and possibly an examination of the cerebro-spinal fluid. Of course the pupillary reactions, and the state of the back of the eye will not be overlooked. Some writers say that knee-jerks are inversely as blood pressure, but this is a very fallible maxim.

[1] See also Spiethoff, *Zentralbl. f. inn. Med.*, 1902.

[2] Mott, F., "Diagnosis of Syphilitic Disease of the Nervous System," *Lancet*, June 12, 1909; and other articles.

Hypochondria, a definite but somewhat rare disease, may present some difficulty; especially if it be associated with defective elimination, and blood pressures instable under nervous, or katabolic disorder.

Hysterical dyspnea.—We are consulted occasionally in cases of dyspnea, really "neurotic," but simulating organic disease; and often in cases of organic dyspnea not due to hyperpiesis, but pulmonary or cardiac in origin. We have seen that in many cases of cardiac disease, in the adjustment of balances, the arterial pressures are raised (see Vol. I. p. 393); in cases of asthma asphyxia may raise blood pressures, as we know (p. 129), and in hysterical or excited panting women the pressures and the heart's action are often exasperated. There ought not to be much difficulty in disentangling such cases as these, but it seems that not infrequently the distinction is missed, even by experienced physicians, and the case interpreted as hyperpiesia, or incipient Bright's disease. The pressure increases are almost wholly systolic. But for fuller discussion of cases of this kind I may venture to refer to my article on Neurasthenia in the second edition of Allbutt and Rolleston's *System*.

The lungs must be watched for any sign of œdema, and suspicions of tissue hydræmia tested by comparisons of the bodily weight. Chronic œdema, gradual in final stages, must be distinguished from acute suffocative œdema.

In some cases it may be difficult to disentangle signs of hyperpiesia from the effects of other heart disease, definite or indefinite. The following case is an example of such a doubt:—

Male, æt. 62. Seen with Dr. Lloyd Jones. Fifteen years before had rheumatic fever, which did some damage to the mitral valve; at any rate there was a mitral murmur; but for some years he had suffered no inconvenience from this defect. Shortly before our consultation, in May 1911, he had been falling off in health, not with cardiac symptoms but with depression of spirits of a morning, irritability, feeling unequal to business, and so forth. He was a very large meat-eater, but said to be temperate in alcohol; however, in answer to closer question, he admitted that he was wont to take a glass of whiskey in a forenoon, as a pick-me-up, though after that he took alcohol with his meals only. No doubt he had lived generously. Dr. Lloyd Jones was called in urgently because of the sudden occurrence of an attack of dyspnea, followed by a persistent

shortness of breath on moving about. Dr. Jones was puzzled at first to find symptoms and signs not consistent with the history of rheumatic mitral disease. From the symptoms and signs indeed it seemed that this defect was slight, and had but little to do with the instant distress. For now the left ventricle was largely hypertrophied, and the arterial pressures very high. The radial artery was thickened and rather tortuous. There was no œdema, no enlargement of the liver, no sign of renal affection, primary or "cardiac."

Of *episodes of high pressures* in persons affected with the decrescent kind of arteriosclerosis I have spoken elsewhere (Vol. I. p. 452); in these cases, if the vessels be thickened, we have to decide whether the state under observation may be one of persistent hyperpiesia with secondary arterial lesion, or of the "senile" form with intercurrent hyperpiesia. Generally speaking, if the previous history be unknown, the results of treatment only will solve this problem: but this test usually suffices: these episodes are remarkably tractable. But cases of high pressures occur every now and then in which the observer catches them in a falling phase, say at 140-150, and assumes them to be about normal; whereas this may be but a temporary phase. Only continued observation can decide upon the course and nature of such a case. The consultant, if he has not the assistance of a family physician who has watched some length of it, may be unable on the data of one visit to give a final opinion.

As concerns the heart itself we must guard against a false interpretation of its embarrassments; and in cases of high pressure we shall not assent to a diagnosis of cardiac inadequacy without asking ourselves—If inadequate, then inadequate to what conditions? Is the organ simply failing in its ordinary duty, or is it succumbing under some abnormal stresses? Is the fault in itself, or in its circumstances? That a heart overburdened by high arterial pressures should fall under the sentence of inherent frailty I have deprecated again and again. For instance, not long ago as I write, I saw an elderly lady with alleged cardiac degeneration; the arterial pressures were high, and the heart was in fact big and labouring on gamely. But the pulse rate was rising to 85-90, a bad sign; and acute œdema was beginning at the base of the right lung, with crepitations nearly as fine as

in pneumonia. This flood continued to rise, and in three more days caused her death. Yet from the beginning the case had been regarded as an intrinsic "heart failure," and treated as such. Such a heart however, if we come to its aid, has a substantial reserve capacity, and often, after the burden is in some measure lightened, responds to digitalis; whereas a heart primarily degenerate does not make much out under any succour.

Concerning "cardiosclerosis" we saw that, unless the case were known to us in its previous course, doubt might arise whether a mitral murmur were due to a primary valvular disease, to a forcing of the valve in a strained ventricle; or again to an atheromatous cusp no longer adapted to its bed. In a middle-aged patient a history of rheumatic fever may be a will-o'-the-wisp. In such a case, one which arises rather in decrescent sclerosis, the murmur is ordinarily not attended with heart symptoms of importance; it does not signify a grave disorder of its functions: more noisy the action may be, but not much less efficient than in atheromatous hearts without murmur. The distinction between a forced leak and a mitral sclerosis must depend much on the history of the case; usually in mitral lesion enlarged the left ventricle is not much altered, and there is no history nor sign of past high pressures. In high pressure cases the aorta is more dilated, and the second sound on the right louder than on the left, whereas in primary mitral disease the converse is the case. Strasburger's method—rate of blood stream, etc.—which is an aid in aortic disease or nephritis, cannot, as he suggests it should, be relied upon in the problem now under discussion. Indeed diagnosis between the big heart of chronic nephritis and that of hyperpiesia cannot be made upon the organ itself; it must be inferred from the other signs and symptoms of the case—the albumin, the persistently low specific gravity of the whole day's urine, the casts, and the uræmic symptoms. It is remarkable that in these big hearts aortic regurgitation is rare, as it is also in the senile disease; though an episode of high pressures may force an atheromatous aortic cusp (see p. 453). While distention of the right heart shifts the "apex" to the left, dilatation of the left ventricle without notable hypertrophy may not materially alter the point of this maximum impulse; the position of the apex may also be

affected by dilatation of the auricles (see also "Heart-swing" Vol. I. p. 396). Professor Waller's estimation by the electrocardiogram of the obliquity of the heart's axis may help us in these distinctions. "Functional" cardiac disturbances are more frequent in hyperpiesia than in mitral disease.

Stenocardial conditions are fully considered in the essays on Aortitis and Angina Pectoris.

Some indecision of diagnosis might arise in *certain alcoholic hearts*. Of the alcoholic heart Dr. Mitchell Bruce says[1] it is always enlarged, say to about 17 oz., and the mitral valve is usually leaky, though there may be no disease of the flaps.[2] There is radial sclerosis in about one-third. The myocardium is degenerate (Mott, Bruce, Maguire). Death is often sudden. Alcoholic nephritis might be confused with arteriosclerosis. However, in my experience there is no difficulty in distinguishing these flabby hearts, really feeble even if they be enlarged, from the fundamentally efficient heart of hyperpiesia, fagged under the strain of inordinate labours. Albuminuria and all degrees of anasarca may be seen in either disease; and in the extremity of either the complete picture of cardiac dropsy, though less commonly in hyperpiesia.

The sometimes difficult diagnosis between *hyperpiesia and chronic renal disease* has been considered at length in more than one chapter of this book. However the difficulty of interpreting some of these cases is well illustrated by this one, which I saw with Dr. Maund of Newmarket:

Male, æt. 61. Is not grey, and looks younger. Made no complaint till four months ago (seen in December 1906), then complained of a vague debility. No headache, no vomiting. Blood pressures not increased, but sclerosis of accessible arteries extraordinarily great. Expression a little blank, and rather staring, but no sugar in the urine. There is a slight cloud of albumin. Heart in normal position and within ordinary limits. No sign of hypertrophy; possibly some diminution, or some emphysema. The arteries were so extraordinarily diseased that one could hardly detect the blood current within. Aortic second sound altered in quality, but not loud. No murmurs. Shortly after our consultation he was seized with convulsions, in which he died. (Uræmic ?)

[1] Bruce, M., *Lettsomian Lect.*, 1901.

[2] See also Maguire, *Trans. Clin. Soc.* vol. xx. p. 235, 1888.

Now what was the diagnosis ? Cystic kidney with rapid and premature decrescent arteriosclerosis occurred to us, but no necropsy was obtained.

In these diseases it may not always be easy to be sure of early degrees of arterial thickening, as the sustained wave, and perchance contracted artery, interfere a little with a due emptying and rolling under the finger ; yet the coats may prove for some time to keep fairly normal. As I have said, in comparatively young persons high pressure may exist for some time without palpable injury to the arterial wall. In Dr. Humphry's case of diseased hypophysis (Vol. I. p. 271) the heart weighed 45 oz., but the aorta appeared to the eye but little altered. The patient's age was 39. The length of the illness was unknown ; he did not seek advice, and was at work up till three weeks before his death. Moreover, as we have seen, a contracted artery is so far a protected artery.

The rule that in renal disease, and in gout, the excretion of purins is delayed, is used by Professor Hopkins as a diagnostic method : all purins are cut out of the diet for three or four days, and then restored ; when, if the kidneys are normal, purins should appear in the urine freely and at once.

Latent œdema may be detected at a very early stage by a rise in weight, when the urine should be tested for salt content ; thus an improving patient may lose weight (see Salt in Diet, p. 93). It is desirable therefore to keep a careful register of the weight of hyperpietic, as of other cardiac and renal patients. In decrescent arteriosclerosis there is not quite the same need for this record.

Epistaxis is no infrequent diagnostic signal of hyperpiesis, as it is of renal disease ; the immediate cause in both being the high arterial pressure. For the same reason it appears not rarely in aortic regurgitation. I have mentioned also that hæmoptysis may have a similar origin.

Healthy persons can generally bear *compression of both carotids* behind the jaw, the vertebrals being of course open, for a few (say ten) seconds ; it has been said that in high arterial pressure this toleration is much reduced, so that in three seconds giddiness and bewilderment appear, and the patient must sit down. I am not sure that the experiment is quite justifiable ; and other

less dubious methods are open to us. In one instance, of a healthy undergraduate, a compression of both carotids produced an epileptic fit; the duration of the compression was brief—how brief I cannot say. The garotte was put on during some jujitsu scuffle. Furthermore, I think that a præternatural susceptibility to this compression exists in other conditions—as for example in neurasthenia.

I have mentioned (Vol. I. p. 162) Dr. MacPhedran's test of the degree of conservation of muscular efficiency in the arteries by the reaction time under nitrites; if the reaction is slight and delayed it is presumed that the quality of this tunic is falling.

Vertigo may be mesophalic, cerebellar, labyrinthine, gastric, anæmic, neurasthenic; Cheyne-Stokes respiration may arise from cardiac, renal, or bulbar disorder; but this last may arise from high pressures. Attacks of spasmodic dyspnea may be due to asthma, hysteria, renal, or cardiac disorder. Hysterical pantings ought not to be misapprehended; they are inconsistent with the other symptoms and with themselves; for instance, the alae nasi are often at rest. In all cases the pupillary reactions and other reflexes will be ascertained, and search made for patches of anæsthesia (hysteria, tabes, etc.).

In **diagnosis of decrescent arteriosclerosis** the outward vessels may be distorted without such disease of the inward vessels as to shorten life, or even to diminish the faculties beyond what is usual in advancing years. And it does not follow that whatsoever maladies may dog the steps of age are due to the vascular decay. Nervous symptoms, such as tinglings, darting pains, numbnesses, cold tricklings, dead fingers, may be simply "functional" and transient; or they may signify arterial obsolescence, or neuritis. For diagnosis then we shall test, so far as we can, the functions of the several organs of the body; searching for traces of albumin or sugar in the urine, for increasing emphysema, latent carcinoma, a growing intolerance of tea, coffee, or tobacco, fatigue on moderate exertion, slurring gait, failing sleep, drowsiness, inability of attention, loss of memory, irritability, depression of spirits, or perchance delusions. Such symptoms, whether directly due to vascular occlusions or to the general drying up of the dews of life—a change cognate with arterial decay—point to biochemical ebbs which, if not

wholly unmanageable, are essentially irremediable. A hæmatemesis (p. 448) may raise an anxious doubt of ulcer, simple or malignant.

An allusion must be made to that terrible disease, as yet wrapped in obscurity, in which arterial trunks are seized by an acute arteritis with agonising pains and gangrene; this "thrombangitis" is not to be confounded with the arteriosclerosis either of hyperpiesis or of decrescence. The senile gangrene of decrescent arteriosclerosis is on the whole painless and belongs to a different series of events. Of this result I have nothing to say which is not well known. Syphilitic arteritis, or chronic meningitis, may be distinguished by headache, vertigo, local nerve lesions—especially about the orbits and face—sluggishness, convulsive phenomena, transient aphasia or other palsies cerebral or spinal, aneurysm, and so forth. Here the Wassermann test, of course, may come to our assistance.

How far is it possible to discern arterial *disease in the inward arteries*? Aortic patches, without giving rise to symptoms, may reach large dimensions, and give warning in the management of life; if, as is asserted, these can be revealed by the X-rays our gain would be considerable. Dilatation of the aorta may be measurable by the same means. Haenisch,[1] in noting this revelation of calcareous patches in the aorta by X-rays, pursued certain experiments which have been made with the rays on the corpse, in the hope of ascertaining the state of the coronary arteries (Simmons[2] and others). So far, they have been unsuccessful; in any case, as Haenisch truly remarks, the practical advantage would be slight, as the existence of calcareous patches in a vessel would not tell us much concerning its permeability. Arnsperger[3] gives a plate significant, as he interprets it, of a calcareous deposit in the aorta; a dark spot is certainly visible at the point signified. The Grödels of Nauheim have given much attention to the indications of the

[1] Haenisch, *Deutsche med. Wochenschr.*, 1912.

[2] Simmons, *Fortschritte d. Röntgenstrahlen*, Bd. xii. Heft 6, S. 371. I have not seen this paper, but quotations from it.

[3] Arnsperger, *Die Röntgen-untersuchungen d. Brustorgane*, 1909. He gives many interesting plates of diseased aortas. See also Williams, New York, Röntgen Rays in *Med.*, 1901.

X-rays in the detection of disease of the walls of the aorta, and with some measure of success. A difference in the two radial pulses would suggest deposit at the forks of the arteries of the neck; on the other hand, a downward swing of the cardiac apex, even to the axillary line, must not be taken too confidently as indicating hypertrophy; we have seen that this may be, and often is, due to a lengthening of the aorta; this in its turn may be due to deterioration of structure, or to the slackness of some kind of atony (Traube). A few records have been published of more superficial trunks thus made visible in sclerosis; but in arteries of the limbs the touch needs no assistance. In so far as thickening of the arteries of the limbs may be any guide to the state of the inner vessels we may detect it early in such vessels as the tibials. Alterations of the temporals seem to have but little significance.[1]

The rate of wave propagation increases as the vessel, whether stiffened by degeneration or made taut and inelastic by sustained blood pressure within, becomes more rigid. I have alluded to this method of measurement, as yet inchoate, in a preceding section (p. 84).

Strubell is of opinion that the electro-cardiogram will prove to be a useful means of diagnosis in arteriosclerosis. His interpretations are formulated in a somewhat too categorical way,[2] and for the present are, in my opinion, in advance of their proofs. It may well be that, with a larger experience of this method, some diagnostic advantages will be obtained. As yet these curves serve best to interpret rhythms; and signify rather the moments and degrees of chemical excitation than those of work done. There is no direct relation between the amount of the excitatory process and that of the correlated work.

Prognosis.—In what has gone before, the principles of prognosis in these diseases, if not formally expressed, are implied. But a few maxims may be emphasised.

In hyperpiesia, when dyspnea has occurred the malady has entered into its last stage, it is hardly remediable, and quite

[1] As regards X-rays in *Phlebosclerosis*, see Carl Beck, *New York Med. Journ.*, April 23, 1904.

[2] Strubell, "Therap. d. Arteriosclerose," *Deut. med. Wochenschr.*, Nov. 7, 1912.

incurable. The case will travel from bad to worse; the heart dilates, it is strung up with digitalis, it gets along again for a while, then relapses, is again perhaps pulled up, and so on; but it is a losing fight. Pulmonary œdema is menacing, and a whiff of it means imminent peril.

In a case of hyperpiesia, of which the symptoms have been dispelled three times only to return, the prospect of substantial amendment is dim.

Hyperpiesis occurring as an episode in elderly persons (p. 453), when it is of course often associated with senile atherosclerosis, is generally curable, and with care to be banished and prevented. Still, when it overrides, however temporarily, a body already frail, we have seen that some harm may be done; an aortic cusp may be forced, an enfeebled heart put to strain, or a corroded cerebral artery burst. It is important therefore to dispel it as soon as possible.

In the earlier stages of hyperpiesia, before the vessels are injured, we may hope to clear the distemper away. What Huchard called "the stage of presclerosis" is, as he said, "very amenable to cure"; the difficulty is to catch the malady in this incipient stage. The French School of to-day speaks hopefully of cure in "hypertendus purs," "sans lesion renale"; if caught in time, that is, before the arterial tree is injured. By one chance or another many patients do present themselves early enough for efficacious treatment, yet too often for their malady only to be still overlooked. It sounds paradoxical to say that the prospect is no better for younger patients than for the older, but one is driven to this opinion. Perhaps, as we see in renal and other diseases, the longer the body remains free from the proclivity the better the resistance to it when it appears; the original bias is less. But the following exemplary case of recovery in youth has been published by Dr. William Russell:[1]

Male, æt. 25. Blood pressure, 220. Although no renal symptom could be elicited, yet the prognosis seemed gloomy. He was put to bed for four weeks on low diet and appropriate remedies. At the end of this time the pressure was down to 135, and after some further courses of treatment it was brought down to the normal. The patient, I gather, did well.

[1] Russell, Wm., *Lancet*, Feb. 13, 1909.

I may add, not of course as proofs, but as illustrative instances, the four cases which follow:

Male, æt. 51. Rather burly, high-coloured man. Health always good, unless "rather gouty"—no podagra. A hearty appetite but no gourmand. Moderate in stimulants. Smokes one cigar a day, and two ounces of tobacco in the week. Heart's apex not much out, but beat hard, and second aortic loud. Arteries normal; and to the finger nothing notable. But the pulse was barely arrested at 200 (Martin's machine). If 10-15 were taken off for attention the pressure remained far too high. Pulse wave sustained. I prescribed the usual diet and regime with calomel, etc. So far as the next few months went he recovered quickly; but one loses sight of people.

Reported by Dr. Edgecombe of Harrogate. Male, æt. 40. Sedentary and a high feeder. Complained of malaise; pulse not obviously tense. Arterial pressures—systolic 190, diastolic 145; apex an inch outside nipple. No murmur. Liver rather full. No symptoms nor signs of renal disease. After a course of diet and spa treatment the pressures had fallen to 170 and 130 respectively. A careful regimen was advised and a return to Harrogate every year. The patient did well, and in four years a permanent cure was established, the pressures remaining at 130 and 100 respectively. The accessible arteries were never thickened.

Reported by Dr. L. Williams.[1] (Rather worse than the preceding.) Male, æt. 60. Diagnosed by an eminent physician as Bright's Disease. Some albumin in urine, but "on repeated examination no casts found, or but a few hyalines. Arterial pressure 200. Heart enlarged. After three months' treatment, including a spa course, the pressures had fallen to 150, and the heart had receded within normal dimensions. No relapse occurred, and he continued to report himself as in good health. A good cure, for that age and with such symptoms. Probably a case in which the causes had been recently active, and the consequences, if acute, yet also recent.

Mrs. B., seen with Dr. Bulmore of Wisbech, January 27, 1909. Female (æt. 60 ?) Depression, fatigue, and broken sleep. Mother died of apoplexy. The pulse had become "extremely tense"; and a rough systolic murmur had appeared over the base of the enlarged heart. Loud ringing second aortic sound. A London consultant of standing had assured her there was no renal disease, confirming Dr. Bulmore's opinion. A few days later I saw her and verified all these points. Apoplexy was feared. It was difficult to stop the radial pulse with the finger, but the arterial wall did not seem much the worse. She was on strict diet and Vichy water, to

[1] Williams, L., *Clin. Journ.*, April 22, 1908.

which we added courses of blue pill. She made a progressive amendment to complete recovery, the pulse becoming soft and normal. I learn that she has continued in good health.

But here we have to formulate the maxim that if the patient can be prevailed upon to obey certain rather rigid rules he may be cured; if not, not. Dr. Mantle [1] speaks of the many patients who present themselves at Harrogate with high pressures, some cardiac hypertrophy and abdominal disorder, but with no clinical evidence of nephritis. For these he has accepted my name of Hyperpiesia, and urges that, if caught early, the condition is curable; if not, it ends in apoplexy or cardiac defeat. He reports instances.

When the malady has gone so far as to have enlarged the heart conspicuously, and impaired the arteries, the outlook is of course so much the worse; indeed complete *restitutio ad integrum* is out of the question. Nevertheless, if dyspnea on exertion has not appeared, something, much perhaps, may be hoped for; the arterial pressures may be mitigated, and the heart may recede even within normal limits. If this can be achieved the impairment of the elasticity of these vessels may not have any very grave consequences; it may mean nothing worse than some circumscription of the sphere of bodily work. Dr. Mantle [2] records a remarkable example of arrest of such a severe case of hyperpiesis. But at this stage the prognosis is far from bright; the arterial system has probably got a new set, and one out of proportion to moderate blood pressures. A radical cure is impossible, and a certain individual standard of high pressure must be accepted; but of course this means a shorter life for the machinery. Notwithstanding, much may be done to alleviate the patient's conditions, so that he may be brought back to a fair semblance of health, and his liability to apoplexy or heart defeat withstood.

The cases of worst prospect are those, at whatever age of adult life, in which vasoconstriction extends far inwards. If not only the radial, but also the brachial and other larger vessels be obstinately tight the prognosis is grave; the mortal issue, whether

[1] Mantle, A., *Lancet*, May 3, 1913.
[2] Mantle, A., *Clin. Journ.*, June 18, 1913.

by the heart or by apoplexy, can hardly be staved off. This condition is most stubborn to treatment; hyperpiesia with radials of normal or expanded diameter—"large leathery vessels"—is more manageable. We shall see that the best course in these cases of extensive constriction is to bleed from the beginning, and to repeat the venesection at proper intervals. Unfortunately, the public prejudices are for the present against this measure, and to persist against a general prejudice may bring other troubles with it; the alarm of the patient and his circle may lead to evasions, and in various ways defeat or hinder our purposes, yet notwithstanding, all these circumstances enter into our prognosis.

One very important consideration I would reiterate, this is, not to rely too much in prognosis upon the range of systolic pressures (Vol. I. p. 90). Besides the emotional and other contingent causes of temporarily exalted pressures, it is possible that normally individuals differ in their blood pressures. I have watched with some anxiety not a few friends or patients whose health is as yet unbroken, but whose blood pressures undoubtedly seemed, all deductions made, to have run high, to 170, or possibly more, even before middle life; and von Basch has alluded to such cases. These persons are of energetic temperament, work hard, and, for many years, with apparent impunity. It is only by chance one happens to discover such subjects. Von Basch had watched one such patient for ten years (up to date of writing), and he had continued in good health. On the other hand, two healthy-looking men, both of whom for many years had worked hard and successfully, but always—as I happened to know—with high blood pressures, both in their seventh decade were smitten with apoplexy. Still to give an anxious prognosis on so chance a discovery would plant an arrow in the heart of an earnest hard-working man, or sensitive woman, and might spoil a life. We fear, but we do not know, that a vascular system working at such pressures must wear out prematurely, and to harass and thwart a life's work on speculative grounds would be very unwise. A man of spirit, were the conditions known to him, would no doubt decide to fulfil the ends of his life in unselfish faith rather than nurse it on terms which would hamper or defeat them; and it seems to me that the physician's place is, after

some cheerful but decisive counsel, to let him live his life as free as possible from apprehensions. Nay, I will go a step farther: let us assume that such early symptoms of high pressures do signify something worse than a quasi-normal idiosyncrasy with perhaps a vascular system of corresponding tenacity; let us suppose that by a free-living or a sedentary life the malady is really on foot; well, it may be, it often is, curable. But in any case, let us not overshadow the man's life by forebodings; let us encourage such an one to rule himself and his conditions in hope and fortitude; for

> If man could see
> The perils and diseases that he elbows,
> Each day he walks a mile; which catch at him,
> Which fall behind and graze him as he passes;
> Then would he know that Life's a single pilgrim
> Fighting unarmed amongst a thousand soldiers.
> It is this infinite invisible
> Which we must learn to know, and yet to scorn.[1]
>
> BEDDOES.

On the other hand, much in prognosis depends upon the amenability and habits of the patient. In some cases we may say, paradoxically, the worse the patient's past habits the better the prognosis; a constitution not yet wrecked may be staggering under folly and error: then, if the patient will be wise and obedient, restoration is often within reach.

In older persons, and in irremediable stages, we shall bear in mind the remarkable insusceptibility of the old to general symptoms, to the acuter general constitutional reactions, so that in old persons especially local signs must be sought and weighed continually and carefully. Of these, I may allude again to any sign of œdema at either pulmonary base; or to a pneumonic consolidation which, even within narrow limits, and unaccompanied by general reaction, may nevertheless prove rapidly mortal. In children, of course, the contrary is the case; in them

[1] Quid tam sollicitis vitam consumimus annis,
Torquemurque metu caecaque cupidine rerum ?
Aeternisque senes curis, dum quaerimus aevum
Perdimus, et nullo votorum fine beati
Victuri agimus semper, nec vivimus unquam ?
MANILIUS (quoted Mackail, *Lat. Lit.*).

turbulent general symptoms may have less sinister meaning, and local disorder be transient.

The chances of lifting on a dilated heart for a while may be estimated by the clearness of the lungs, and by the response to alteratives and digitalis.

At the risk of being tedious, I must here again insist that in making a forecast of the issue of a case of arteriosclerosis, my distinction between the hyperpietic and the decrescent mode must be borne continually in mind, as must also any suspicion of renal or other toxic condition. Persistently low diurnal specific gravity of the urine is of ill omen. Under what circumstances hyperpiesia may be curable or incurable we have discussed already at length ; it is a serious malady. Decrescent arteriosclerosis is variable in its incidence ; in some cases it is associated with only too evident signs of the graver atrophies, with loss of facial expression, shuffling gait, lapses of memory, and so forth. In other cases the vascular decay seems to pursue the visceral arteries ; the aortic and mitral valves degenerate, and the great vessels decay, while the mental functions are but little, and the muscular not gravely, impaired. These patients may die of asystole, the hyperpietic of dropsy. In others again the decrescent disease proves consistent with length of years and a persistence of no inconsiderable abilities, whether of mind or body. One guesses that in these cases the incidence is chiefly upon the long arteries of the limbs, but, if so, these decay in the easy classes as in labourers. I have still many a friend whose vessels, so far as they are in touch, are, and for many a year have been, grotesquely deformed ; yet who still turn out as fair a day's work as most of their contemporaries, if with a slower recovery from fatigue. The limitation of powers in such persons often lies less in the quality of the work than in endurance, and more and more in fatigue after effort. The labourer is incapable of a full day's labour every day, and so is liable to dismissal ; but, in shorter hours and without bodily exertion, the parson, or the judge, may still turn out excellent work. Dr. Mitchell Bruce has said (*loc. cit.*), " I have known a man live from the age of 73, when I first saw him, and then found his radials sclerosed, and a systolic murmur in the aortic area and albumin in his urine, to the age of

93, enjoying reasonably good health." (This was, no doubt, a case of my decrescent sclerosis; no hyperpietic could show such a history.) Dr. George Oliver writes:[1] "Hypertonus is a frequent and menacing concomitant of arteriosclerosis. . . . Arteriosclerosis without high pressures (*i.e.* my decrescent form) is consistent with length of days and a vigorous and active old age, providing that the blood pressures are moderate." They are moderate; the truth is, the cases are of different nature from first to last, and the prognosis depends upon whether the patient has the one disease or the other. Sir Thistleton Dyer once told me that Sir Joseph Hooker, at least thirty years before our conversation, had presented atheroma of the radial arteries, and evidence of vascular disease elsewhere; he ceased all exacting work and retired to the country, and was then in good health, with all his faculties unimpaired, at the age of 91, "though his arteries were worse than ever." I do not feel by any means sure that in these cases we are to assume too readily that the sclerosis is wholly peripheral, and that the visceral vessels are intact. The post-mortem revelations are by no means consistently to this effect; hearts and brains which during life had shown little sign of failure, have after death exhibited vessels in states even of well-marked decay. Happily the vessels of the base and the great ganglia of the mid-brain usually harden before those of the cortex; when those of the cortex suffer, the mind must sink in dilapidation.

Concerning Life Assurance, I have collected some materials but find myself unable from them to formulate any maxims of service. The tables furnished fail to discriminate between the sclerosis of high pressures and the decrescent mode.[2] So long as Arteriosclerosis is accepted as a "disease," statistics are impossible; the tables are compiled on heterogeneous materials; or, if blood pressures are taken, only the systolic are recorded.[3]

[1] Oliver, G., *Clin. Journ.*, 1908.

[2] *E.g.* Burwinkel, *Zeitschr. f. Versich. Med.*, 1909, Nos. 6 and 7. Dr. Janeway's laborious survey (*Arch. of Int. Med.* vol. xii., 1913) of many cases of high blood pressure labours under another confusion, a confusion of all kinds of morbid processes attended with such pressures. He was well aware of it, but did not see his way to disentangle the coil.

[3] *E.g.* the returns of the North Western Mutual Company of the United States from 500 districts, city and rural, in which their referees are using the sphygmomanometer. Under tests for life insurance few systolic pressures would represent the mean condition.

No doubt a rule to reject all persons of 40 years and upwards, whose blood pressure (systolic) ranges above a certain figure, will exclude many persons unfit for insurance ; but, on the other hand, it would exclude many more whose claim to insurance would be unjustly denied. So long as systolic pressures only are registered, the fallacies, as we have seen at length, are manifold ; but, unless it be by the auscultatory method, which unfortunately is a subjective record, we are not yet ready, either with instruments efficient enough, or knowledge mature enough, to undertake the test of blood pressures as a condition of insurability.

CHAPTER X

TREATMENT OF ARTERIAL DISEASE

Arteriosclerosis then, Bright's Disease apart, is, as we have seen, not a clinical but a pathological name. The arterial tree, as a whole or in parts, may undergo injury or deterioration in the course of more than one series of morbid events; we have seen that however closely in the advanced stages all sclerosed vessels may resemble each other in their superficial, or even in their histological aspects, the systemic disorders on which they ensue may be several. If, towards the end of their life, the arteries abut upon a common form of decay, the processes of initiation are nevertheless various, each with its own primary pathological features. For arteriosclerosis itself there can scarcely be any treatment; but as the modes of disorder leading to such damage or decay of the arteries are several, each must have its own way, and its own means, of therapeutical aid. It is in default of this discernment that many careful and serious chapters on the therapeutics are in confusion;[1] arteriosclerosis and high blood pressure are taken as convertible terms, and as constituting a single malady. But, as I must pursue that discrimination of the kinds of arterial disease which I have adopted, let us consider the treatment of:

(1) Arteriosclerosis, the effect of persistently high blood pressures (Hyperpiesia).

(2) Arteriosclerosis (Toxic), the effect of certain poisons or toxins as of syphilis, typhoid fever, lead, diabetes, etc., in many of which the blood pressure is not necessarily, or usually, much increased, if at all; although in others, such as plumbism, it may be raised throughout.

[1] *E.g.* Strubell, *Deutsche med. Wochenschr.*, Nov. 7, 1912, who classifies on superficial, not radical, features.

(3) Arteriosclerosis (Decrescent), the effect of "senile" involutionary changes, in which form again the arterial pressures, if increased, do not exceed the quasi-normal increase which is general in later life; unless indeed hyperpiesis intervene, as not infrequently it does (Vol. I. p. 452).

It is evident, then, that if these several processes arise in different ways, they must be counteracted on different methods.

In the first variety **(Hyperpiesia)** the increase of peripheral resistance, upon which the rise of pressure may depend, is of obscure origin. "The immediate cause is increased friction, which must depend either on a narrowing of the arterial bed, or on an increase of viscosity of the blood; or, of course, upon a combination of these factors." [1] How the viscosity of the blood varies we have seen; on the other hand, we have seen also that the arteries may contract persistently over areas large enough to maintain a considerable rise in the general pressure. The compensatory mechanisms may be liable to get out of gear, or become "labile." The proximate cause of such arterial constriction may come from without, may be a product of distempered metabolism, or may be an intoxication by refluent waste; we have seen that some such poisons exist, poisons which may act upon the vasomotor centre, or widely upon the vessels themselves. Until we understand these conditions rational treatment must be retarded.

If we can catch hyperpiesia early, and keep at work against it, it can be cured more often than not. "Malum nascens facile opprimitur; inveteratum fit robustius." Before, then, a systematic treatment of hyperpiesia is commenced, we must ascertain what, in the individual case, it is possible to achieve. When the system has taken a new set, the whole has readjusted itself to the altered conditions, and the new attitude is more or less permanent. Therefore, to bring back the old equilibrium of pressures by active treatment is out of the question; and diligently to attempt it is to do more harm than good. Our first business must be to satisfy ourselves, as far as may be, of the stage at which the patient

[1] I am quoting my own paper of 1894. See recently Martinet, A., *Pressions art. et viscosité sanguine,* Paris, 1912.

has arrived. If, in a comparatively young man, say of 45-50, after a period of bed and simple treatment, the heart has receded within its normal dimensions, if the pulse has softened, and the aortic second sound is abated, we may suppose that the disorder had not been long agate, and that the system may not yet have suffered strain, that it may not yet have got that new set. In such persons again and again we see that *restitutio ad integrum* is quite possible. But if the vessels have begun to show signs of strain, and the heart remains persistently out of bounds, a bias has arisen which thenceforward may have, more or less, to be accepted. If the morbid stresses could be removed altogether, the altered bent of the arterial tree might never be rectified; strained vessels cannot be made anew. Notwithstanding, some compromise may still be practicable; if return to the normal form be unattainable, by gradually modifying the stresses the system may have so much resilience left as to find an intermediate resolution. These conditions cannot be apprehended all at once; for a while our observations must be vigilant and continuous, and therapeutic measures kept below the activity which makes the patient feel uncomfortable. Here the subjective sensations of the patient are a useful guide.

Furthermore, I must reiterate (see Prognosis, p. 76) the precaution pertaining to all therapeutical methods, but which bears with peculiar weight upon Hyperpiesia, lest by our diligence and counsels we awaken apprehensions and meticulous self-examinations of a kind and degree injurious to the patient's equanimity. We should be slow therefore to order any long stoppage or suspension of business, lest he "sicken of his vacuity," which Milton said was Hobson's fate when his business was broken up by the plague of 1630–31. We have seen that a vigorous middle-aged man, when told that in spite of a seeming of health he is the subject of abnormally high arterial pressures, due probably in their turn to some perverted state of his nutrition, may fall into a dread of an apoplexy, or of heart defeat. He will be watching himself, wondering how his pressures are running, touching his pulse every hour or two, deliberating on his diet, fearing to walk up hill or stairs, until he becomes hypochondriacal. In many cases, much, even to the goal of recovery, may be done, and a forecast of favourable issue given in

a cheerful and hopeful spirit. And I repeat that, in spite of our reasonable desire to trace the periods of the arterial tides, we may have to deny ourselves the advantage of frequent observations and, when made, we shall be wise to keep the records to ourselves. By the finger and the stethoscope a fair notion of the patient's phases can be got: and if we are always silent about figures, whether good or bad, the patient ceases to ask about them, and abides content with general appreciations. We shall tell him truly that as the arterial pressures are very unstable, and our instruments rough, we are guided, not by the gauge only, but by broader views of his conditions as a whole. I have learned that, fallacious as under the patient's anxiety they are, to make incessant pressure observations as tests of progress is unwise. Having got a grip of the case and of its principles, we shall pursue our reasonable methods with as little fidget over details as possible; so long, that is, as the patient is in no instant peril.

Nevertheless the proper preaching has to be done; it is idle to begin a course of treatment so prolonged and systematic as these maladies require unless the patient has will enough and perseverance enough to submit to tiresome rules, and to methods which will make no little claim on his faith, so long may it be before their good effects become plainly manifest and abiding. Let him retire from posts of vexation and fatigue, whether in business or in society; and let us impress upon him the usual if rather futile advice about "not worrying"; but let us not all at once knock a man off all his duties, either temporarily or permanently. However for most patients, it is true, we have to establish new habits; to persuade the man to relinquish the customs and manners of a lifetime. Some tractable persons accept our counsels kindly, and pursue them methodically; but unfortunately the majority of our patients are of other temper: some are impulsive, wilful, intolerant of discipline, especially of self-discipline; others, if more reasonable, are of ardent, untamable natures, admirable, it may be, in all relations of life except those of the invalid; others again imperiously demand at the hands of the physician a cure they will not try patiently to compass for themselves. Patience, gentle persuasion, sympathy, a sense of humour and knowledge of men may do much, and

convert even the rebel and the faithless. For, as Huchard well said, the question is one not of a drug but of a regime.

The foundation of all treatment must lie in the resetting of the natural conditions of the patient, and in "Naturheilkunde" *diet* (Vol. I. p. 238) comes first. *In the cases of children* (p. 172) the rules of treatment, empirical as they are, are promptly efficacious. The diet must be restricted, particularly in respect of fats, sugars, meat, and strong broths. In these cases even milk may be given too liberally, especially in its natural state; for a child of 10 years of age, thus disordered, one pint of milk, prepared in one way or another, may be sufficient for the twenty-four hours. Indeed the quantities of food must be moderated in all directions, for the mother is too apt to stuff the child, or the school-boy to stuff himself. During the ailment, three or four rusks and a cup of milk are enough for breakfast; a little light broth, with a biscuit, may be given in the forenoon, and at dinner some plain white fish, or chicken, with but little potato, and a light pudding. Full plates of farinaceous or stodgy puddings, flatulent and clogging to the digestion, are to be forbidden; light steamed bread puddings, a little blancmange, junket, and the like, will be preferred. Tea to be as breakfast, rusks or dry toast being better than thick slices of bread. Butter is to be given scantily; fruit, raisins, currants, jam, cakes, not at all. Some milk food will make a sufficient supper. The temperature in these cases sometimes rises a little in an irregular way; when this is so, the food should be even more sparing for the time, and but little of it solid. If feverish the patient must stop in bed; otherwise he is better about, gentle exercise out of doors being encouraged.

Of medicines, mercury is the chief. It may be administered as calomel or as grey powder. Grey powder is the form of which in children I have most experience. Under the use of fractional doses the breath sweetens, the actions of the bowels become more normal, and the stools less offensive. For a child of 10, a quarter of a grain of grey powder thrice daily may be ordered, and this in repeated courses of four or five days at a time. The mouth must be closely watched, and kept very clean. If the drug produces two motions a day, we need not hold our hand; but if mucus becomes evident in them, the remedy should be reduced or suspended. As the tongue cleans, and the other symptoms

are mitigated, a bitter stomachic mixture may be administered. Children dislike bitters, but they are efficacious, and if pleasantly flavoured will be taken by well-disciplined children. They should be given before the meals. The diet may now be cautiously enlarged, but fats, sweets, and even starches are to be given with a sparing hand. These children are often of nervous stock, and their ailments may be coloured by whims and ill-humours ; still, the disorder seems substantially to be one of the *primae viae*.[1] When thoroughly clear of the disorder, it is helpful to give these patients cod-liver oil ; during the colder months of the year this fat is so well digested by the young that it may displace with advantage some of the other "rich" elements of the dietary. Under this kind of management the full sustained pulse and obtrusive artery gradually subside. When the vessel returns to its normal tenuity I cannot tell, as before this comes about one loses sight of the patient ; but, if some years later an opportunity arises for a re-examination, it may be found, should the amendment have been maintained, that the walls of the vessels have returned to their normal state (Vol. I. p. 178). The variation may lie in an increase of the muscular tissue only. Whether these children are more apt than others to fall into the hyperpietic disorders of later life, I am not yet able to say. This is one variety of arterial disorder ; there are others for which we have as yet no explanation, and therefore no rule of treatment. Latent infections may be concerned in them.

In *hyperpiesia of the adult*, the symptoms may be not unlike. Unfortunately, as we have seen, hyperpiesis often establishes itself in the adult without betraying its presence, so apt is the bodily system in adjusting itself to abnormal conditions. Indeed high pressure in the cerebral vessels may give rise, for a time, to a sense of well-being. A man of middle life may report himself to be in excellent health, when, for a skin eruption perhaps, or for life insurance, he comes to a physician, who may discover a systolic pressure of 160-190 mm. Hg. The radial artery may be already a little thickened, and the left heart moderately enlarged, yet it may still be possible to restore the health more or less completely if the man, who had regarded himself as healthy, will

[1] Some recent observers have made the interesting suggestion that these cases may prove to be mild degrees of acidosis.

submit on trust to the long, close, and vexatious medical treatment, and the irksome management of his diet and habits, of which I have spoken. Diet is the foundation of all treatment; Hecht[1] says definitely that by diet hyperpiesis is reduced, "in vielen Fällen bis zur Norm," in many cases cured. Strauss, Hoffmann, and our own physicians give the same testimony. On the other hand, to continue in this disorder for a few years more will lead to death by apoplexy or by heart failure, even if life be not cut short sooner by an acute pneumonia.

The patient then may get rid of his plethora and his cardio-arterial strain, if he will pay the price. But I cannot help repeating that at a somewhat later stage the outlook is not so hopeful; the circulatory tree—heart and vessels—is now permanently altered, and a restoration of the normal balance may be no longer practicable. In such a state to try to reduce the arterial pressures to the normal disturbs the artificial balance without re-establishing the original mean, and the dietetic and other treatment is not completely successful; at best it is a compromise; yet the compromise, in moderate cases, may suffice to keep the disorder at bay, to postpone or prevent an apoplexy, and to husband the resources of the heart.

Happily, if my argument is sound, the heart in these cases is usually of good quality; its coronary circulation, till checked by atheroma, is at high pressure; and even in atheroma, if the progress of the disease be gradual, the nourishment of the heart, stretched and strained as the organ may be, is kept up, somehow or other, with a wonderful steadfastness (p. 542). The so-called "weak" heart is often staggering under systolic pressures of 200 mm. Hg, and great bulks of blood! As an old writer says, "After a plentiful supper, and his usual load of liquor, he complained; etc. etc." Our treatment of "weakness" of this kind must not be that of a heart, under ordinary or low pressures, failing intrinsically. Enlarged such hearts certainly are, and sooner or later, if an apoplexy be averted, will suffer defeat; but we have seen that commonly such overdriven hearts hold on heroically. With caution, and at certain moments, the physician must use such cardiac stimulants as digitalis or strophanthus; yet his main duty is not so much to spur the heart

[1] Hecht, *Zeitschr. f. klin. Med.* Bd. lxxvi. Hte. 1 and 2.

on as *to relieve it of its heavy burden.* For, even if recovery of the normal adjustment be past hope, much may be done to maintain for a while some measure of equilibrium.

In stages of high pressure, early and late, the dietetic cure is in principle the same. In many instances the rise of arterial pressure is due to excess of food, positive or relative; but whether the harm is done by accumulation of putrefying stuff in the bowels, or overcharge of the excretory organs by a profusion of digested but unused food in the circulation, is not known, and ought not to be taken as known. We do not know how far luxurious feeding means merely a passage of excess by the stool, or a taking of food into the blood, only to be excreted; or again an incorporation in the tissues with inadequate incorporation of oxygen, or of constructive or destructive enzymes. The subjects of this plethora, large eaters as they may be, may not be fat, nor ruddy; often they are so, but not a few are lean and sallow. In some cases indeed the intake has not been more than many persons dispose of easily—individuals vary widely in capacity for disposing of excess of food—but has been more than the individual capacity. Not a few big feeders, thanks perhaps to good liver or kidney, manage to attain longevity. Many of those who suffer, whether stout and ruddy or lean and sallow, if they do not present a personal history of gout, yet come of gouty stock, and are wont to regard themselves and their people as gouty. Moreover, treatment directed against the supposed gouty habit, especially a spa treatment, often answers to expectation. If the man be fat, he must gradually reduce his intake till he brings himself back near to the weight of his earlier years—say to his weight at the age of 40. In men of or beyond middle age, to *bring the food down gradually*, from the old habit of vigorous youth, even to half the quantity habitual to the patient, has in many cases the happiest results. Sir William Osler says that confinement to bed for a couple of months during severer reduction of the feeding is efficient in moderating pressure; a precaution to which I shall return. In a certain case—one of aneurysm, treated on strict Tufnell diet and bed—Cushing[1] noted that the blood pressure fell from a morbid height to normal, and there remained for two months. If

[1] C. Cushing, *Boston Med. and Surg. Journ.*, March 1905.

alcohol alone does not lead to atheroma (Vol. I. p. 249), yet in conjunction with other causes — as we may see with lead-poisoning—it has a strong contributory influence. The same may be true of tobacco. Alcohol therefore must be cut out, or, lest we fall into "un certain snobisme d'hygiène," as a French vintner said of teetotalism, reduced to a nominal amount; and tobacco, tea, and coffee must be strictly moderated, especially if there be any arrhythmia. Some persons find in whey a pleasant and harmless drink. To "lactate" drinks I will refer presently.

As regards the chief classes of foods—special dietaries—temperance rather than exclusiveness should be observed, especially, it is supposed, in respect of the nitrogenous, and of those which contain purins; but really of the effects of purins we know little, and in most cases the main purpose is the restriction of the whole intake. Erasistratus, who paid much attention to plethora, preferred strict moderation of diet, and periods of starvation, to venesection; but he used some kind of compression of the vessels of which we have no precise information. A monotonous dietary, which may conveniently lessen for the gourmand the temptations of the table, may for the temperate and tractable patient blunt the appetite to some inanition. Those abstemious persons who are so little able to manage, normally and completely, even moderate quantities of food that their habit fails to avert, or to dissipate, pressures morbidly high, unfortunately for their virtue, have less promise; the original bias lies deeper (Vol. I. p. 241). But some of these patients, such as well-to-do ladies and closet students, are also of sedentary habits, and their excretory functions are sluggish; for them ordinary quantities of food are relatively excessive, and restriction of food and alteratives may suffice for cure.

As regards particular foods, what are we to say when Brault and Huchard, both of great authority in this subject, say, the one (Huchard) that meat is poison to the hyperpietic, the other that, as atheroma occurs in the herbivora, a vegetarian diet is fraught with this mischief! In all these cases the purin-containing foods should be closely watched, and, so far as the patient's tastes and appetites permit, reduced, yet again and again I have placed high-pressure patients on purin-free diets

(Walker Hall, and Haig), or on vegetarian diet with cheese, milk and eggs, with no appreciable reduction of blood pressure within such limit of weeks as to satisfy the conditions of an experiment. Surely, if the purins were the cause of their increase, the pressures should have fallen notably within a week or two. Because a food is poor in purin we have assumed in the past—we may have erred in doing so—that it would not affect the general purin metabolism; we have yet to learn whether this be so or not. However, let us suppose that the less purin the better, and that all animal soups, gravies, and other meat extractives should be forbidden. We should forbid also high meats, such as game and salmon, potted meats, salt fish, caviare, and all "made dishes." Potain used to assert that in a case of chronic renal disease a cup of beef-tea (*bouillon de bœuf*) would send up the pressure by 40-50 mm.; but this might have been due to the salt in it. Dr. Oliver advises that, in order to reduce extractives, the meat of the dietary should be boiled. There is no practical difference between white meats and "butcher's" meat, but as red meat is more sapid more of it is eaten. The "Salisbury" diet, of mince-meat and hot water, certainly seems to be remarkably efficacious in some of these quasi-gouty cases, purins or no purins.[1] But we are not to overdo even abstinence.

For the following description of a purin-free dietary I am indebted to an anonymous article in a recent number of the *British Medical Journal*;[2] as it expresses my own experience I give it in summary:

After a probationary period the "*purin-free*" *diet* is thus prescribed. The term "purin-free" is however a misnomer. A precisely "purin-free" diet would be composed of milk, eggs, white bread, cheese, butter, and a few vegetables; but when the milk contains many cells, as often it does, and the eggs are not quite fresh, as often they are not, their purin content comes within the range of our present chemical methods. However, to return to a dietary designedly poor in purin. For this purpose the following foods are at our disposal: Milk—fresh or soured, buttermilk, or whey; eggs—boiled,

[1] On vegetable diet and viscosity see Vol. I. p. 117.

[2] I have mislaid the date, but probably in 1911 or 1912.

poached, scrambled, or raw; white (not brown) bread and butter; macaroni and cheese; rice, tapioca, semolina, and vermicelli. Suet may be used for puddings of all kinds, such as currant or jam roll, treacle, apple dumpling, etc. Pastries, pancakes, jellies, and the usual tea cakes are also available. All vegetables except the pulses (peas, beans, and lentils) are poor in purin. Practically all fruits may be permitted. As to drinks, tea, coffee, or cocoa are excluded; hot water, claret or burgundy, mineral waters, or hot milk may be substituted. Beer and porter should be discontinued, and indeed all alcohol diminished as far as the usual habits make possible.

After a month or so of this diet some return to customary food should be attempted. Brown bread, oatmeal, wholemeal, beans, peas, nuts, asparagus, or mushrooms may be gradually added; and if it is desired to employ vegetable protein only, these should play a large part in the dietary. When animal protein is permitted, sweetbread should be the first kind of meat allowed, since the purins contained in sweetbread are "bound purins," and are hardly absorbed, the greater part passing out in the fæces. Later, codfish, sole, plaice, mutton, chicken may be permitted; salmon, halibut, and beef and pork being reserved for better days. When meats are taken they should be stewed, not roasted; and milk sauce substituted for the meat gravies.

Even when a return to ordinary diet is made we should still restrict tea, coffee, cocoa, and soups. With regard to extracts, there is little to choose between any of the familiar preparations, all are to be avoided. The purins of yeast extracts are less well absorbed than those of meat extracts, therefore presumably less harmful; but the differences are slight.

But I would not have it forgotten that the prescription of a diet poor in purin calls for vigilance on the part of the physician. He will watch for the danger signals of starvation, of glycosuria, and even of mental bias, while some careful return towards ordinary diet should be aimed at. We may gain something by the daily determination of the total purin output or, when this is impossible, the weight and general condition of the patient may be a sufficient guide. Under this regime there

will probably be a diminution of the amount of protein consumed, but, as the quantity of food necessary for the maintenance of life and for the provision of latent resistance to disease varies with every individual, and with the same individual at different periods of life, so it is well to realise that prescriptions and rules as to food can rarely be applied rigidly; the attainment of good results from the use of a diet poor in purin depends not a little upon the sagacity of the physician; even then I have found it not so successful as enthusiasts suggest.

Milk diet is extolled by some writers as helpful if not curative; some say that it substitutes a harmless for a poisonous bowel content, others that it stimulates the thyroid gland to work in abatement of blood pressures.[1] A milk diet, exclusively speaking, can be but a temporary resource—say for a few days; a drinkable bulk of it contains too little food for a continuous diet (4 litres = 2·300 cal.), unless the patient be kept in bed. If we desire to withhold lime, the milk must be decalcified with sodium citrate. In any case, rice, eggs, and sound cheese, foods which give nutritive value without bringing in purins (Bradford), or even a little meat, must soon be added to it.

The *Karell cure* consists not in the milk only, but also in Oertel's method of relief of the heart's work (not of the blood pressures) by reduction of fluid. Many plethorics are copious bibbers, and bring on "polydipsia" and "polyuria"; a perverse equilibrium. The fluid in the "Karell cure" is cut down to 1-1½ litre, the milk being given in four doses, at 8 A.M., 12 noon, and 4 and 8 P.M. After two or three days some egg, biscuit, and flour are added. This diet should not be pushed beyond ten days. Even if the urine be scanty and thick, under the lesser intake diuresis will set in; and on the spare nourishment blood pressures should fall, and probably viscosity also, unless the patient were hydræmic. Much harm is done at spas by reckless gulping of waters, under the quackish pretension of "washing out the uric acid"; for all the time, especially if the kidneys be inadequate, the patient may be hydræmic. This, and such conditions, must be distinguished from that of essential cardiac

[1] Chalmers Watson, *Lancet*, Oct. 12, 1907, and *Brit. Med. Journ.*, Dec. 21, 1907, who thinks "there is no special therapeutical effect in a purin-free diet, not equally attained by a carefully planned diet without such special reference."

failure, in which the Karell and Oertel plan must be followed with great caution, if at all. We must not forget that milk is not by any means a saltless diet; but of this I shall speak presently.

A strictly *vegetarian diet* is bulky, and low in proteid value. Oliver thinks that under it high pressures are moderated. I have been disappointed with all these narrow dietaries—the Haig diet, the vegetarian diet, and so forth; patients do feel, it is true, marvellously better, even for some months; they are relieved of the effects of food too rich or abundant for them; but in six months or a year they revolt. Not only is the new diet insipid but they find it inadequate. Yet as temporary expedients these exclusive diets, if used with care, are efficacious. It is a common belief, which I share, that oatmeal is a valuable and wholesome food deplorably neglected. It is difficult nowadays to get good oatmeal; the advertised oatmeals are too often adulterated with inferior cereals, and the meal of the best oats is spoilt if ground and bolted on the modern rollers and silks. To be nutritious and palatable oatmeal should be stone ground from the best oats. Dr. William Russell says that oatmeal, supplemented by such booty as they could lay hands upon, was not only the domestic but also the commissariat staff of life of the Border raiders. A vegetarian diet is, of course, rich in lime, a condition of arteriosclerosis which I have already discussed (Vol. I. p. 504). An anticalcareous diet, such as Rumpf and others prescribe in arteriosclerosis, would mean also the exclusion, or great reduction, of milk. But this mode of degeneration will always find lime enough; even if calcification were mischievous, what Ludwig Braun calls the "naïve practice of withdrawal of lime from the food," is absurd. But calcium salts might perhaps so increase the viscosity of the blood (Vol. I. p. 110) as to raise blood pressure, or determine a thrombosis. As to the sum of protein required per day, or per week, for the permanent support of the body, the opinions of experts differ so widely that we must take it as unknown. We are pretty sure however that it is considerably less than was supposed in the last generation. Still my experience is that ultimately under an insipid or monotonous diet the appetite flags, the digestive reactions grow languid, the spirits sink, and any ill proclivity of the

system is perhaps promoted, or at any rate not withstood. A good deal of allowance has to be made also for old habits and individual constitution. A philosopher, whether medically qualified or no I forget, has said: "Nut cutlets, proteid potatoes, lentil sausages, mock forcemeat, vegetable sauce, uric-acid-free marmalade, malted wheat biscuits, sterilised cream cheese, non-caffeine coffee, apple juice wine: Result, severe domestic discord!" It is as a temporary expedient at a time of crisis that a limited or even an ascetic diet, while working with the patient's hopes, may be, as often it is, the most effective of all our therapeutical means.

Fruit, as I have said, is a welcome and usually wholesome food for subjects of cardio-arterial disease, save those who are disturbed by dyspepsia, distension of stomach and bowels, gripes, or lax stools. Dr. Jerome of Oxford found that, so far as they agreed with the individual digestion, pears, fresh figs, grapes, dates and oranges might be taken, even by gouty persons, with impunity, and even with advantage. Apples and pears, apples especially, agree better when cooked; mastication is rarely an efficient pulper of a crisp apple.

Tea and coffee, which contain purins, are less and less well tolerated as years advance into age; and thus may carry some significance as tests of normal circulatory reactions. But concerning them, and similarly of *tobacco*, I have written at length on another page.

Antiseptic measures, addressed directly to the bowel and given by the mouth, are probably ineffectual. If such an effect be urgently needed, β-naphthol is, in my experience, the best of these drugs; the sulphocarbolates may have some value.

Concerning *common salt*, we have seen in former chapters that there is much to be said; and in this respect a distinction between hyperpietic, renal, decrescent and toxic arterial disease is essential. Some writers say that salt tends to raise blood pressures, but as yet on insufficient evidence;[1] much must depend on the rate of its elimination in particular cases. Dr. Leonard Williams says also—what however is not quite the same proposition—that a reduction of salt intake may abate high pressures. The part of the

[1] See Boyer, *Journ. Exp. Path. et Pharm.*, July 1907.

kidney in these ionic balances is still obscure. In hyperpiesia the kidneys may be intact; moreover kidneys in disease may for a short time still let through certain bodies, caffeine for instance, with no less freedom, even if taken in excess. In the third stage of hyperpiesia however, when the heart is getting beaten ("Hochdruckstauung"), the congested kidneys fail duly to discharge the salt, and a fortnight's salt discipline may be needed. Otherwise a day or two of deprivation now and then will suffice. During salt abstinence a little lemon or tarragon will make the food more sapid. A kidney which is detaining sodium chloride may pass methylene blue, but it is a long jump from this uncertain ground to Ambard and Beaujard's conclusion,[1] when they assume that plus blood pressure means plus salt ("chloruration"). They infer that, if digitalis cause a diuresis of salt, then digitalis is a "hypotensive" drug, and suggest that theobromine, which has many advocates, may often compass the same end. Achard, from another point of view, thinks that salt is retained as a protection against some toxin; other writers say that the toxalbumins retain the salt, whereby pressures are raised. All these conjectures are still in the clouds; but we are more and more sure that somehow or other sodium chloride may be so retained in the body as to cause, or to be correlated with, œdema manifest or latent (Widal's "pre-œdema"). Vaquez prescribes complete salt deprivation for two days the first week, for one day in following weeks; the whole salt intake on intermediate days not exceeding 1½ gm. of salt per diem. If, he says, in a hyperpietic case we notice cough, distended stomach, mental depression, dyspnea or heart disorder, withdraw the salt. How, by watching body weight, "pre-œdema" may be ascertained I have said already. Whatever the explanation may prove to be, the effect of withdrawal of salt from the diet in œdema is often amazing; and if the uncertainty of this result hitherto shows that we have not yet grasped the full conditions of the problem, yet the dissipation of serosities often clears up menacing conditions, even cerebral symptoms, in a surprising way. Any saline ingredients in the medicines must of course be taken into the reckoning. Salted meats, often also indigestible in texture, must be forbidden, and the salt-cellar

[1] Ambard et Beaujard, *Arch. gén. de méd.*, mars 1904.

used sparingly if at all. Whether then the salt be held up in the tissues, or accumulate behind defective kidneys, we do not yet know, at any rate not in particular cases. We must watch the bodily weight, and manage the salt accordingly. On the other hand we shall beware lest we run salt deprivation too hard. If at a time of urgency all salt must be withheld, even in the baking of the bread, this rigid exclusion should not last more than a very few days, if only because of the languor of appetite and digestion which may ensue.

That there is any harm in other spices and condiments, which may be grateful to the palate, I cannot say; but "highly spiced" food usually means something more, it means rich and elaborately dressed dishes. In dressings lemon juice must be preferred to vinegar.

While we were all engaged in lightening the protein cargo, and filling up with starches, sugars, and fats, we were pounced upon by Dr. Francis Hare, who urged that *the carbohydrate extreme* was at least as perilous, if not more so. We shall go at least this far with Dr. Hare, to admit that in a being so complex as man, whose body has been developed and balanced during ages untold by an infinite variety of external conditions, either, or any, extreme rule of life is to be avoided. We know by direct experiment, particularly by experiments with large doses of syrup, that normal men differ widely in their relative capacities for this function and for that, and particularly for carbohydrates. It is the delicate office of the physician to equilibrate, so far as his science can tell him, the natural capacities of the individual patient and the sum of his conditions. Senator [1] also, although he dreaded nitrogenous putrefaction in the bowel, has cautioned us against excess of carbohydrates in hyperpiesia, especially in fat people; he preferred to give them a little meat with fruit and green vegetables. He prescribed also gymnastics and light purgation. Dr. Graham Steell [2] prescribes a diet much on the same lines as those of Senator's later papers. I think that, although overfeeding with carbohydrates may be more harmful, and the excess not so readily burned up as we have supposed, yet the harm of excess of proteid,

[1] Senator, *Ther. d. Gegenwart*, März 1907.
[2] Steell, G., *Med. Chron.*, Dec. 1902.

of which we need comparatively little, must be far greater, except in persons whose capacity of dealing with carbohydrates is constitutionally narrow.

Although Chittenden's researches suggest that mental work makes a remarkably small demand on food, yet unfortunately sedentary mental work in towns seems to sharpen the appetite. The standard of the real needs of the body must, more or less, be regulated by the scales; but in spare patients who are not large feeders we must be guided by the progress of the case, and by clinical observation.

To turn now to the issue of the food; the abiding high pressure suggests to us that metabolism is imperfect or perverse, that waste proteids are re-absorbed, or that certain organs are in defect. Although it is an error to suppose that an annual six weeks' "cure" can take the place of a systematic, patient, and persistent regimen, yet from spa treatment, as for instance at such resorts as Harrogate, Royat, Evian, Carlsbad, Marienbad, Homburg, we shall see (p. 103) that striking results are to be obtained.

It is in the first, often latent, stages of the malady, in sound subjects, when the abnormal pressures are not of long standing, and the consequent ill effects upon the heart and vessels are incipient, or even yet to come, that cures are to be made. Periods of five years or more, according to morbid degree and individual equation, may elapse without permanent strain. In the first and curable stage, unless the peripheral resistance be unusually high and obstinate, there are better methods for the restoration of the circulation than artificial baths and exercises; namely, *natural exercises*. In such cases—and we should now be catching them earlier and earlier—patients are to be encouraged cautiously to take more and more exercise. As Thomas Adams said, "Labour and moderate diet are the poor man's friends, and preserve him from the acquaintance of Master Doctor, or the surfeited bills of his apothecary." When the heart has been relieved of stress, and is pulling itself together, gentle games may be recommended, especially such as do not require sudden efforts. Gentle cycling on the level, quiet walking, even on the lower hills, golf, and so forth, are useful. The efforts of tennis, even of the lawn variety, are too sudden. It is of enormous advantage if we can throw

open the vast muscular areas to the blood currents, and thus lower resistance and wash away impurities. Then, if all goes well, for patients disposed to recurrent hyperpiesis but in whom the vessels betray as yet no considerable signs of lesion, there is no medicine like carefully regulated hill-climbing. This "terrain cure" for rising blood pressure is provided, together with other appropriate means, in some English health resorts, as at Church Stretton. The purpose of the method needs little explanation. When a healthy man essays to climb a hill, he will find, as I have taught for many years, that during the first two or three minutes the radial artery maintains its calibre; so probably do the chief arterial beds in the skin and muscles. During this period, the exertion raises systolic and diastolic pressure, embarrasses the breathing, and throws more stress upon the heart. But on cautiously proceeding, although systolic pressure may still be high, and even rise, the radial artery dilates, not gradually but rather suddenly, to perhaps twice its lumen, and the pulse becomes dicrotic; this signifies, no doubt, the opening of other large arterial areas also; therewith diastolic pressure falls, and the respiration gains freedom; we get, as the phrase goes, our "second wind." During the initial effort no therapeutical caution can be too great; but after this expansion the walker will proceed with more and more ease. In an untrained man this expansion comes later, and in a hyperpietic later still. Until it occurs, the patient should proceed very cautiously and slowly in his ascent. Indeed this expansion coefficient should be our guide in regulating the exertion. The patient may soon learn to observe it. Arterial (radial) expansion is more important than pulse rate; and a few extra systoles during this period need cause no alarm; if the exercise can properly be continued these will disappear. Even in healthy but untrained men some care in hill-climbing is necessary at first, but it is hill-climbing especially which is effective in developing the respiration. Potain made blood-pressure observations on three classes of recruits: (i.) new recruits, (ii.) recruits after six weeks' training, (iii.) recruits of nine months or more standing. All were between the ages of 20 and 24. In those of the second class, the blood pressures fell permanently by an average of 10 mm.; in those of the third it ranged 20 mm. lower. In their gymnastic teacher it was

30 mm. less than these, observations which agree with Dr. Michell's on undergraduates.[1]

How far this searching but efficient method may be applicable to a particular case must depend on the discernment of the wise and expert physician. Every person from the age, let us say of 45, would be wise to have the arterial pressure measured every four or five years ; oftener, if it be found above the mean for the period of life, or if morning depression of spirits, disturbed sleep, constipation, biliousness, or other vague discomforts suggest that the exchanges and discharges of the body are going wrong. Upon him who comes of a family in which gout or apoplexy has appeared, such counsels are the more incumbent. The muscular and fascial stresses forward the blood by pressure, if, indeed, they do not exert some suction upon it (Braune quoted by Krehl), and the peripheral vessels dilate. The effect of muscular activity on the circulation was well illustrated in the days of bleeding, when, if the stream did not run freely, the operator would request the patient to clasp and unclasp his hand quickly. The muscular system is not an organ of locomotion only ; it is also a hearth of heat generation, and probably an organ of metabolism.

In exercise, natural in early and mild cases, or in severer or more critical cases artificial, *the respiration* is of course a large factor. Although, if exercise be begun gradually, no healthy temperate man, if 50, 60, or even 70 years of age, should feel short of breath on climbing hills, at the mountaineer's pace, yet step by step his breathing becomes deeper.[2] Whether chiefly by washing out carbon dioxide, or by carrying in oxygen, or again by the " respiratory pump " acting upon the circulation, as exercise proceeds the mean arterial pressures fall, and the heart, pulling itself together, diminishes in diameter (de la Camp, and subsequent orthodiagraphists), and viscosity is reduced. The mere inhalation of oxygen or of carbon dioxide while at rest has no obvious effect.[3] Unless in cyanosis or anæmia, conditions

[1] *Vide* my art. " Heart Stress," Allbutt and Rolleston's *System*.

[2] I need only refer to the well-known observations of Haldane and Smith, of Pembrey, Oertel, Beddard, Keith, Halls-Dally, Hill and Flack, G. N. Stewart, and others. Also my " Heart Stress " article (*loc. cit.*).

[3] Stewart, G. N., *Journ. Pharm. and Exp. Ther.* vol. ii., 1911, and *Journ. Exper. Med.* vol. xiv., 1911. But see also Hill and Flack, *Journ. of Physiol.*, 1910, vol. xl.

in which natural exercise would not be prescribed, or at any rate not hill-climbing, the bulb has probably less to do with the benefit than the muscular activities, skeletal and respiratory. The respiratory machine Miura of Tokio calls the second heart; it may however be added, as a third, to the peripheral heart, which also in hyperpiesia is disordered. Miura says that as in enlarged heart the inspiratory muscles are hypertrophied, so in enlarged left heart are the expiratory. In passive exercise he massages and faradises both sets of muscles.

Familiar as the warning has become, I ought not to omit to repeat that towards middle life it is foolish, in the midst of a sedentary life, to try to redress the balance of function by plunging into unwonted muscular exertion. After long inaction the peripheral vessels do not respond quickly to sudden demands; and after the age of 36 the elastic limits of our tissues are narrower. If the town worker, even as a comparatively young man, is to return from his holiday with a purified and invigorated body, he must train himself gradually to efforts which a few years before he had found more promptly possible and beneficial. For a definite task it is calculated that the trained man can get done by 12 per cent of his possible energy output what in the untrained takes 30 per cent of it. In middle life such efforts bring more weariness, and exhaust the animal spirits, at any rate in the first seven or ten days of the holiday. The recent fashion of golf, if not a manly game for young men, is nevertheless an admirable pursuit for men of or near middle age and after; for it can be taken easily or more energetically as the player's state of training may dictate. It is difficult to speak with patience of the men who, in the intervals between their feeds, lounge in motor-cars. Riding on horseback, the best of all exercises for adults, seems, except in the hunting-field, to be falling more and more into disuse; and with this growing indolence and luxury blood pressures will be augmented. Let us remember then that with gluttons, as indeed with all well-fed sedentary people, we must begin exercises very gradually and watchfully, and not forget that a fall of pressure may be due less to purification than to fatigue. We shall make ourselves sure also that the heart and vessels are still sound: no systolic murmur at the base of the heart; no bronchitis or emphysema to increase cyanotic viscosity.

But let us suppose, always in respect of hyperpiesia, for of it, not of the decrescent disease, we are still speaking, that we cannot thus develop our exercises from quiet walking to regulated hill-climbing: how are we to act? The heart is doing all it can; the vessels are not to be trusted, or the patient is elderly. Well, we must go to the other extreme, and put the patient to bed, looking forward to advancement by passive exercises, and by baths. In bed, say for a week or ten days, excessive arterial pressures usually fall slowly, sometimes near to normal; probably because of opening of the periphery by the equable warmth. In a week or ten days we learn what in the individual can be done in this way; the pressures may reach some lower but still persistently high level. Of course other factors, such as pulse rate, output, cardiac signs, and so forth, must be carefully observed; a fall of pressure may be due to declining heart.

This plan of bed for a few days answers very well for the episodical cases of hyperpiesia in elderly persons (Vol. I. p. 452), but in no case should it be continued for more than (say) a fortnight without passive exercises; and he must be a very sick man who needs a longer rest in bed, even during artificial exercises.

Of the artificial exercises, for cases in which natural exercises are inappropriate, *massage* is perhaps the best. Much depends upon the skill of the massor, especially in manipulation of the abdomen, a very important part of the reaction. In massage of the limbs, Dr. Herbert French lays stress on expert kneading and stroking of the arteries themselves, whereby a better nutrition of their coats may be obtained (or a morbid constriction reduced (MacWilliam)). Thus the peripheral or, by abdominal massage, splanchnic areas may be opened out, the movements of blood, juices and excretions quickened, and viscosities reduced. Dr. French speaks of a "stiffening" stage of the muscular fibres, which later, unless averted by massage of the tunics, will end in fibrosis. Whether the arterial muscle gets out of training, and thereupon stiffens, I do not know. Oberndorfer [1] also thinks that by the massage of ordinary exercise the peripheral arteries supplying the joints become less prone to sclerosis. This direct method seems to be more applicable in decrescent sclerosis than to arteries under hyperpietic

[1] Oberndorfer, *Deutsche Arch. f. klin. Med.* Bd. cii.

stresses. In this kind, the way to save them is indirectly by moderating the high blood pressures, to which end also massage is a valuable aid. The long vessels of the limbs, almost the only vessels which massage can reach directly, may, as it seems at present, fall into one or other mode of arteriosclerosis ; the ordinary mode in which the media perishes by the encroachment of the intimal change upon it, or the mode, or degree, known as Mönckeberg's (Vol. I. p. 482), a medial necrosis issuing in calcification ; a dissolution which may or may not be associated with a substitutive fibrosis partly from the intima. In this mode however fibrosis is not a primary or a prominent feature. Fibrosis, due to lateral pressures, mechanical stress, is apt rather to arise under the influence of high blood pressures, primarily in the intima (Thoma, etc. ; see Vol. I. p. 489); but in the arteriosclerosis of high pressures (Hyperpiesis) the median muscle can scarcely be languishing for lack of work. It is in this condition that large arterial areas may be over active, spasmodically constricted, even in the limbs, as indeed we may often perceive in the radials. These problems cannot then be discussed fruitfully unless the distinction between hyperpietic and decrescent or senile arteriosclerosis be continually kept in mind ; for in at least half the cases of sclerosis there can be no fibrosis due to exorbitant pressures. These reflections are by no means intended to diminish the value of Dr. French's method, which will probably prove very useful both in the decrescent and in the hyperpietic kinds ; if it be not required for the vessels, it is well adapted to open out peripheral areas such as the musculocutaneous, and to reduce resistance. Massage of the limbs and back seems normally to cause a rise of arterial pressure, at any rate at first ; of the abdomen, a general fall ; but if there be a wide splanchnic constriction, this natural reciprocation may not follow. Miura's massage and faradism of the muscles of respiration, and the value of " respiratory gymnastics," I have already mentioned. Respiratory exercises should be practised gently, the patient being first encouraged to draw deep breaths. It is said that high arterial pressures may be reduced by breathing in rarefied air ; of this method I have no experience.

The systematic *passive exercises of Lang* and August Schott for cardiac dilatation have no special place in the treatment

of any kind of high blood pressure or arteriosclerosis, or in hyperpiesis not until the heart is suffering; then these methods, with other means of cardiac therapeutics, may take a secondary place. Otherwise, both for mind and body, voluntary exercises are to be preferred. In hyperpiesia the "Nauheim methods" are not generally required; the heart, if it can have fair play, is good enough; nevertheless, in occasional cases, after reducing the mean blood pressures, baths, exercises and digitalis may assist us in helping the heart back to more normal diameters. Gibson preferred the Swedish to Schott's method.[1]

Emphysema of the lungs, frequent in the decrescent class, in hyperpiesis would add to our difficulties. When the patient has begun to suffer from cardiac dyspnea, and dilatation is advancing towards mitral incompetency, we must recognise the unwelcome truth that the heart is undergoing defeat; a substantial and permanent restoration of cardio-arterial balance is not then to be hoped for; on the contrary, fall of pressure by heart-failure may be feared. Then the pulse usually rises in rate. Something may yet be done, but too many leaves have been torn out of the book.

On *climate* in hyperpiesis I have little to add to the dictates of common sense. In high-pressure cases the patient should not exceed altitudes of 2000-2500 ft., and these heights should be reached cautiously; it is better to state 1500 as the limit. Mountains, and northern and eastern aspects, should be avoided. Two or three times patients have informed me that it was while stopping at high altitudes that the symptoms of high pressure first manifested themselves, especially as dyspnea, chiefly nocturnal; and thrice at least under my own observation subjects of hyperpiesia have been seized at high elevations (4000-5000 ft.) with apoplexy. Too sudden a change from low to high altitudes raises arterial pressures (Potain, and others since).[2] And as statistics seem to indicate that apoplexy is more frequent in cold weather, we must take precautions accordingly. Nevertheless, vigorous and undamaged subjects of early hyperpiesia, who can be gradually inured to moderate heights, and to

[1] Gibson, G. A., Section of Pharm. and Ther. Brit. Med. Assoc., 1912 (*vide Brit. Med. Journ.*, Aug. 17, 1912).

[2] See also Oliver, G., *Lancet*, June 13, 1903. But see Vol. I. pp. 181-2.

hill walking, find in the sweating during exercise in the dry evaporating air of the Alps a remarkable effect for good. There are of course other contingencies to be taken into account, such as the vasoconstricting effects of cold to the skin.

For decrescent arteriosclerosis a mild and equable climate at moderate elevations is to be preferred from the beginning.

We read of sanatoriums in America where all these methods, with proper diet and medicines, are co-ordinated and variously administered according to the needs of particular cases. Dr. Sprigg has, I believe, opened such an Institute near Banff. In companies people will submit to a drill which singly they would shirk.

Diaphoretic methods, at first sight likely to be useful in high pressure, are, in my experience, disappointing. I have tried hot-air baths, such as the "Turkish," hopefully, carefully, and perseveringly; but they have not given relief to the patient (often, indeed, they have caused discomfort), nor established any abiding reductions of blood pressure. Now I do not advise them.

Of *spa baths* there is something more to be said, though it is not easy, in the absence of any discrimination between the arteriosclerosis of one series and of another, to discern the significance of most of the reports of bath treatment. Moreover the sphere of medical experience at a spa is confined to such cases as it may be customary to send thither. Notwithstanding, we may clear the problem to some extent by putting aside, at any rate for the present, decrescent arteriosclerosis, for which baths are of no specific service, and keeping our attention upon excessive blood pressure, a condition over which we might expect baths to have some influence. In cases of this series however von Basch, whose experience at Marienbad was very large, found no advantage from baths; indeed he thought they often did harm. Dr. Oliver, on the other hand, from his Harrogate experience, has recommended the Aix douche-massage, followed by a needle bath of alternating temperatures, and warm pack. Dr. Mantle of Harrogate says that hyperpiesia is quite curable there if taken in time.[1] My observation is that in a bath of 98-100° F. the radial crimps up for two or three minutes, during

[1] Mantle, *Lancet*, May 6, 1911, p. 1206. A useful article on many points. See also "Thermal Environment and the Circulation," by Dr. Lyth, 1913—an attempt to reduce these problems to scientific method.

which time it is difficult to catch a record ; then the height of the pulse-wave rises, and the mean pressure falls 15-20 mm. Pawlow, I see, has demonstrated this fall, and its persistence for some time after a warm bath. The initial rise of pressure should be avoided. It was no doubt in the second phase that Schapals, with the ray screen, saw the heart diminished in size, and found the arterial pressures not increased.[1] We may anticipate, I think, that the douche-massage and warm pack, in expert hands, would coax the peripheral circulation into activity, and with the alternating needle-bath would re-create a fairly equable vasomotor system. What is certain is that bath temperatures—whether of hot air or hot water—above or below the "neutral zone" drive up the arterial pressures, not for the moment only ; and albumin, as in too cool a bath, may appear in the urine. It is clear that we must begin at the neutral zone, and cautiously increase the temperature as the periphery opens out. Dr. Fortescue Fox,[2] for the release of vasoconstriction and high pressures, advises "subthermal" baths, at a temperature between those of the blood and of the surface, and with these and even cooler baths he tries to keep these conditions at bay. He recommends, very reasonably, prolonged baths, as at Leuk, in natural warm waters below blood heat—say 89-90° F. Thus any initial rise of pressure sinks to a small proportion of the whole effect. Ottfried Müller, who has made some careful observations on this subject,[3] says all douches are "cardio-gymnastics," as they raise the blood pressure, and this the more if above or below the indifferent point ; it seems desirable therefore to begin with the warm pack, and follow very cautiously with "vasomotor gymnastics." A warm bath alone leaves the skin susceptible to chill ; and Winternitz declares that friction with water at 40° C. and 8-10° C. alternately, carried out vigilantly and scientifically by experts, not only prevents the cutaneous susceptibility, but also lowers the blood pressure on the whole. This method may be beneficial to persons essentially sound, such as early non-renal hyperpietics whose heart and

[1] Schapals, *Zeitschr. f. exp. Path. und Ther.* Bd. x., 1912, an interesting article on baths at various temperatures, etc.

[2] Fox, F., *Brit. Med. Journ.*, Feb. 15, 1908.

[3] Müller, O., *Deutsche Arch. f. klin. Med.* vol. lxxiv., 1902, p. 316.

vessels are still equal to such discipline. Such methods have another kind of good effect also, a bracing effect upon the nervous system. Müller, besides the effect of douches, came to further conclusions: that water baths a little under the indifferent zone (say 33-34° C.) raise blood pressure, not momentarily only but during the whole bath, and retard the pulse; that a little warmer, and gradually warmed up to (say) 40° C., they cause a momentary rise of pressure, but then a considerable fall, even near normal; then comes a renewed rise, which is persistent: that baths over 40° C. cause a persistent rise, but with plus pulse-rate; that "*Nauheim baths*" raise pressure more by the temperature than by the carbonic acid gas, but both affect the pulse (if the Nauheim bath lowers the blood pressure great vigilance is needed); and, finally, that all sweating baths—hot air or steam—raise both blood pressure and pulse rate. Groedel says confidently that by careful precautions rise of blood pressure can be avoided. But nearly all these baths affect the heart-work, and the greatest care is needed in cases in which the heart is either overborne or degenerated, or in which the kidneys are behindhand. Pulsus alternans would be a bar to bath treatment, and to all "vascular gymnastics." The function of the kidneys is to be tested by the diuresis method, as at Royat, Evian, etc.; by phthalein, or, if possible, by Ambard's method (Vol. I. p. 322). Ortner agrees on the whole with Müller.[1] "Indifference" in a bath—plain, saline, or effervescent—seems to be an ideal point, or one would say: Begin at the point of indifference so as not to start vasoconstriction; then with care raise the temperature, opening out the surface vessels gradually. The advantage of saline, or of a mantle of bubbles upon the skin, is that a cool bath does not feel chilly, and that changes of temperature are moderated. In Luftperlbäder (bubble-baths) some authors think it does not much matter, save for suggestive purposes, what air is used—common air, CO_2, or O; but it is better to keep to common air, as the temperature conductivity of gases varies considerably, and is not parallel with the water temperatures. Moreover air bubbles are a little less adhesive to the skin than those of CO_2,

[1] See also article by Sadger (of Wien-Gräfenberg), "Die Hydrotherapie der Arteriosklerose," in *Ther. d. Gegenwart*, Nov. 1908.

and so in critical cases are less stimulating. H. Senator and Schütgen[1] find that air and oxygen bubble-baths are less irritant than those with CO_2, and that at indifferent temperatures they lower blood pressures. They relate two cases of air bubble-baths which lowered pressures more than did warm baths, CO_2 baths or O baths. Hydropathic systems are not carried out at home with the same expert supervision and scientific method as in Germany, too much is left to the attendants; so, as German ways of life are unsuited to the habits of most of our countrymen, my experience of hydropathy is not very large. During the bath or pack, at any rate in new patients, frequent observations of pressure and of rate should be taken; there must be no cooling down; warm wrappings, and at least an hour's bed, should follow every bath.

Dr. Groedel,[2] who has studied the effects of Nauheim baths with discernment between hyperpiesia and decrescent arteriosclerosis, although he claims so complete a control over their pressure effects, wisely advises great caution in their use in cases of high blood pressure or of stenocardial attacks; though with care they may be available. Unfortunately after the bath the blood pressure rises again rapidly, an oscillation which in stenocardia may be distressing and even injurious. Begin, he says, as closely as possible to the temperature of the skin, and with very little gas or saline. It may be judicious at first to give only partial baths, and of short duration; though it would seem well, if some preliminary rise of pressure cannot be avoided, to carry the bath into the second stage, that of fall of pressures. Peat baths, with careful grading of temperatures, are said to be effectual in lowering blood pressure and accelerating the blood stream through the periphery; this I am ready to believe, and their warmth is easily kept constant.

However English physicians have carried out a few careful observations on both methods; Drs. Edgecombe and Bain,[3] for instance, have published a series upon eleven adults; and to Dr. Mantle's observations I have referred. The Harrogate

[1] Senator, H., u. Schütgen, *Deutsche med. Wochenschr.*, 1909, No. 35.

[2] In many papers; one of the latest in *New York Med. Journ.*, March 15, 1913.

[3] *Journ. of Physiol.* vol. xxiv. No. 1, 1899; and *Lancet*, June 10, 1899.

conclusions did not quite correspond with those of Müller, Ortner, and Groedel; but they indicated likewise that on cool immersion arterial pressure, both maximum and minimum, rose considerably, while venous pressure fell; though after the reaction this effect passed off. The effect was still more marked when the percussion of the needle-bath was added. In these experiments hot immersion and dry heat lowered both arterial and venous pressures in the second stage, and might double the output per second. Warm saline baths lowered arterial pressures more than did plain water at the same temperature. Superheated air baths are forbidden by all the experts. Dry massage of the trunk and limbs lowered the arterial pressure, but abdominal massage, by driving the blood from the splanchnic area, raised the general arterial pressure. The Aix douche lowered the general pressure (see p. 103). These various conclusions point to great complexity in the problems with which we have to deal, and to the need of further careful observations; the evidence, as Schapals says, is very conflicting. It is said that in arteriosclerosis the vascular response to baths is more sluggish. However, unless the transient rises of pressure be too sharp, or the vessels too frail for any risks, we may be well rewarded if cardiac balance, output, peripheral activity, better elimination, and a net fall of pressure be obtained. The permanent result may be an all-round amendment due to the cumulative effect of many coefficients; but it is in the milder cases, and in persons under middle age and of fair vascular condition, that such risks may be safely taken. That we must distinguish the momentary from the permanent effect of baths on the blood pressure is clear; but in critical cases a momentary effect may be all-important.

In England, where the bathing is generally left to attendants, these scientific niceties are not observed; it is better therefore in this country to treat arteriosclerotic patients, whether hyperpietic or decrescent, by other methods; for baths are not indispensable.

As regards the *drinking of waters*, we find no less conflict of opinions. Ortner says that hot Carlsbad water raises arterial pressures; other physicians declare it has a lowering effect. Bourgouignan,[1] a pupil of Huchard, affirms that the Evian

[1] Bourgouignan (*loc. cit.*).

cure (and Vittel and other springs are probably as efficacious) doubles the output of urea, triples the output of uric acid, and increases the chlorides (on which he lays much stress) by one half. Dr. Leonard Williams gives a like report of it, and adds that it does not lose its virtues by export, so that it may be used at home. If the water is not so quickly absorbed as to produce prompt urination, the difficulty usually lies in the stomach or bowel; therefore during the digestion of the water abdominal massage is ordered, and hot applications are placed upon the belly; or the kidneys may be at fault, if so, warm baths and tepid drinks are employed; or, thirdly, the delay may be due to some obscurer conditions of the system, to be met by cutting down the quantities of all other liquids, and administering the Evian water in small doses to the patient on bed or couch. Bourbon-Lancy is strongly recommended by French physicians; but the choice of the spa and the course of the cure must be adapted to the individual case. Of the use of the Marienbad waters, especially for persons of gross habit, we have abundant testimony in von Basch's writings; his results do not differ much from those now generally accepted. Yet, to tell the truth, I am reluctant to pour large volumes of fluid, fluid which may contain common salt, into the bowels of persons with high pressures. Their ingestion must add to the work the heart has to do, and dispose to that latent œdema which, as we have seen, is so apt to impair the elasticity of the peripheral circulation (Vol. I. p. 38). Vaquez,[1] I see, has taken up this problem. In the normal state, he says, the kidney responds by adequate functional activity to the ingestion of 600 cc. of water taken in a morning before food; but more readily if the patient lies down. In renal inadequacy the discharge is much slower and may only be fulfilled, if it be fulfilled, during the night; and this only if no further dose be taken: then, if day by day some undischarged remnant is not got rid of, an accumulation gathers for mischief. Thus when, in arteriosclerosis of any form, the renal values are in doubt, exact observations must be made for a few days before spa waters are used freely.

Waters containing radium emanations, as at Bath, Kreuznach and elsewhere, are now in much request, and are credited with

[1] See, *e.g.*, Vaquez, *Arch. des mal. du cœur*, avril 1913.

virtues which, as yet reported, rest on very unsubstantial evidence. Plesch, no mean authority, says that under careful use of radium waters both maximum and minimum pressures fall, and metabolism is rectified. The supposition is that they greatly increase diuresis, excretion of uric acid, and exhalation of carbonic acid; that they lower blood pressure, and diminish blood viscosity. They are not to be used in "Granular kidney." It is claimed of course that the natural radium waters are far more efficacious than the artificial. The natural bath should be given about the indifferent temperature, and should never last longer than twenty minutes. Radium emanation has been used by many physicians in the treatment of high blood pressure, and while some of them urge caution, and note some unpleasant effects, all seem disposed to admit that it has some influence in reducing pressures, and perhaps in diminishing the viscosity of the blood. Dr. Lowe [1] of Bath has discussed the arguments on both sides of the question.

Light baths, a system of incandescent electric lamps giving off indeed both heat and light, were recommended by Huchard and others, and have made some way since. If the lamps be turned on suddenly, the blood pressure of the patient rises; the rule therefore is to bring the lamps gradually forward from 60° to 80° F., and then, if no ill effect, to increase the heat from 85° to 100° F.; at this point both maximum and minimum pressures are said to fall, and the heart to empty itself more rapidly and completely. This amelioration is said to last for some hours. But baths again can be thus used only for persons whose cardio-arterial system is not undermined. That they act by way of reducing adrenal activity is one of those many guesses which obscure the discussion of these subjects.

Electric Treatment.—Faradic baths, cutaneous stimulants, such as saline or gaseous baths, or fresh air, are of no service in hyperpiesia. Reports of the therapeutic value of high-frequency currents in cases of high blood pressure are not very consistent, and some are contradictory. My own impression of the method, and I have used it in many cases, is decidedly favourable. In three cases, which I was able to watch for three or four

[1] Lowe, T. P., *Brit. Med. Journ.*, April 20, 1912.

years, one of them under Dr. Roberts of Harrogate, the not inconsiderable reduction of pressure at each sitting persisted for two or three days or more, and proved to be the forerunner of an abiding amendment; although in each patient the malady, being only too well established, ultimately returned. In another case, a lady under the care of Dr. Philpot of Chester Square, Dr. Philpot told me that at the first sitting the pressure (over 160) fell a little, but fell still further afterwards. After the second sitting it fell to 133. This fall after the sitting I have observed in other instances. It is very important therefore that after the sitting the patient should remain at rest. Dr. Sloan of Glasgow,[1] who has used the method for more than two years, finds it effective in high-pressure cases; it lowers peripheral resistance, and must not be used in low-pressure cases, as the reduction might be harmful. He advises a rest before as well as after the sitting. Dr. Sloan considers it proved that the current influences the alimentary canal; and he adds a conjecture that it modifies metabolism and dissipates toxic substances. Dr. Somerville [2] agrees with this suggestion, and emphasises also the influence of the current in relieving insomnia, and promoting renal excretion. One may hazard guesses of this kind but, although Dr. Somerville alludes to some stricter researches of his own, so far as I know, no systematic experimental proofs are produced as yet. It seems at least as likely that a diathermic effect upon the vaso-motor nervous system relaxes arteriolar spasm (Lewis Jones).[3] Dr. Somerville, whose conclusion was based upon 266 clinical cases, says indeed that among the after-effects is a rise of the surface temperature of the body.

Some authors suspect—Kraus, for instance, who seems rather sceptical of this method [4]—that the fall is due to suggestion. One can scarcely suppose suggestion to act without a concept in the patient's mind; and a concept of falling blood pressure can scarcely be formed; all we know of expectant attention in this matter is in the direction of a rise of pressure. Kraus admits however that by the current the pressure is lowered, even

[1] Sloan, *Brit. Med. Journ.*, 1907 and 1910; see also Sayer Ettie, *Brit. Med. Journ.*, 1910.

[2] Somerville, W. F., *Lancet*, Aug. 6, 1910.

[3] Jones, L., *Lancet*, Feb. 7, 1914.

[4] Kraus, *Berl. med. Gesellsch.*, Feb. 24, 1909.

in chronic nephritis; also that the effect is more or less abiding, and no harm comes of it. Dr. Oliver who, when in Harrogate, had found no help in hyperpiesis from other modes of electrical treatment, contrasted them with the high-frequency current, which on the contrary he had often found effectual. A Zürich physician, Bühler,[1] observed the good effect of the high-frequency current, even in severe cases; the falls were decisive, and after six or seven sittings persistent. He attributed the effect, at any rate in part, to considerable (*deutlich*) fall in viscosity, which he says he has verified. All his patients have assured him that they have suffered no disadvantage from it. His best results were in early cases of high pressure. From the Zürich school also I have obtained a Dissertation (of 1911) by A. Antonjewitsch on "Arteriosclerosis and High-Frequency Current." The author works in the usual confusion between high blood pressures and any arteriosclerosis, but testifies to the potency of high-frequency current in moderating high pressures; he regards it as the most efficient of our means. He found that the nervous symptoms and insomnia also were often relieved. Normal and subnormal arterial pressures were left unaltered by it. That the current is usually harmless is true, perhaps true of all cases in which it has been used carefully; in skilled hands it is without danger; but Dr. Sloan's warning is echoed by other physicians. In a discussion at the French Medical Association of 1906, Doumer and Chanoz stated that by "Arsonvalisation" they found abnormally high pressures to be reduced; and with their experience other speakers were in agreement, and I have heard at second-hand that Vaquez and other French physicians are using the remedy with success. Schurig[2] says of it that "above all things it has a favourable influence upon arteriosclerosis (*sic*)." The method is probably quite impotent to dissipate any kind of arteriosclerosis; but the author evidently means high pressures, for he proceeds inconsistently to say that it is especially useful in "presclerosis," gradually reducing the arterial pressures, and therewith the associated symptoms, such as headache, vertigo and insomnia; that it quickly restores energy and good spirits, and lifts up the general well-being. His report is perhaps a little too

[1] Bühler, A., *Schweiz. Korresp.-Bl.* No. 13, 1911.
[2] Schurig, *Deutsche med. Wochenschr.*, Feb. 6, 1913.

sanguine; yet many clinical observers speak in like manner. After these words were written, Dr. F. H. Humphris read an important paper to the same effect before the British Medical Association in 1913.[1] The author, who evidently writes with considerable experience and technical skill, protested against the objection that the treatment of hyperpiesia by high-frequency currents was disappointing; that if so, either the choice of the case or the technique was at fault. Too often, he said, the current used (d'Arsonval) was limited to 250-300 ma., whereas it should run at 1000 ma. The sitting should last for 15 minutes. A pleasant feeling of warmth and comfort then pervades the body, with somnolence. The blood pressure should fall 20-40; say from 200 to 180 or 160. Towards the following day it will gradually rise, but not always to its original point, when another sitting should be given; after a time it will be observed that the pressure no longer rises between the sittings; but to keep the pressures about this range will require further sittings, from time to time as the course of the case may indicate. Dr. Humphris wisely did not pretend to explain the good effect, but guessed at vasomotor relaxation, and "improved metabolism." He stated indeed that the solids of the urine and perspiration were increased, but gave no detail of analyses, nor references. If the effect be directly and only vasomotor, presumably some further treatment would be required, but the author thinks that, by setting the peripheral circulation free, metabolism is thereby freed also, and a vicious circle cut. He spoke also in favourable terms of the influence of the electric light cabinet.

On the other hand, Carrière[2] warns us that in one case, in which the pressure stood at 260, it was reduced by the current to 160 (!); but the urine fell too, asystole set in, and in four hours alarming symptoms of dyspnea, convulsive spasms, and acute pulmonary œdema. Happily the crisis was surmounted. Probably this was a renal case, and we have seen that in chronic renal cases this method is to be used cautiously, if at all. Martinet gives us the same caution; indeed he says plainly that in albuminuria with imperfect renal excretion it is contra-

[1] Humphris, F. H., *Brit. Med. Journ.*, Oct. 11, 1913, p. 935.
[2] Carrière, *Paris méd.*, 1912, 191-194.

indicated. It seems then that in this agent, if carefully used, we have a potent means for good, if ill-managed, for harm. The doses should be exactly measured, and the sittings timed; the first few sittings should be tentative. As with the bath a brusque start would jerk up the pressures. To persons whose pressures are disposed to fall, it is said to be unpleasant. The patient should feel warmer and better after it, the peripheral circulation being so expanded as sometimes to cause a limb to swell, as in hot weather. In elderly persons with fagged heart it should be applied meticulously, and for decrescent arteriosclerosis, even if the pressures should range 150-160, the method is neither indicated nor effectual; in no case can I think it desirable to reduce the pressures by 100 mm., or even by 50 mm., as is not infrequently recorded or advised.

I have said more than once that to mistake early stages of hyperpiesia for neurasthenia is no infrequent error in diagnosis, and it is chiefly in these early cases, or in some of them, that the high-frequency current may prove to be permanently effectual.[1] The reports are still rather inconsistent; in some cases, cases probably of advanced stages, the reduction of pressure may fail to do any good. However, if in particular instances[2] it has had its failures and disappointments, especially in the earlier attempts, yet there is a strong current of opinion in favour of high-frequency treatment of non-renal hyperpiesia, especially in earlier stages. Fortunately it is a remedy whose doses can be measured, and the effects appreciated, with some accuracy.

It is not unlikely that the effects of the current are due, not to electric, but to diathermic action; this question the therapeutist may leave to the experts.

No other form of electricity is of any service in hyperpiesia. The effects of the *sinusoidal current* have been carefully investigated by Albert Weil and Mongeot.[3] It raises pressure, not by stimulating the heart, but by constricting the periphery, an effect which could play only a subordinate part, as a "vasomotor gymnastic," for which purpose graduated baths or gaseous douches are more convenient and manageable.

[1] See, *e.g.*, Macnamara, *Lancet*, July 18, 1908.

[2] See Guilleminot, *Arch. d'élect. méd.*, Aug. 25, 1911. I have seen only the column of abstract in *Epit. Brit. Med. Journ.*, Jan. 20, 1912.

[3] Weil and Mongeot, *Bull. Therap.*, 1906.

At the Peebles Hydropathic Institute Dr. Luke tells me that he is getting good results in hyperpiesia by "*rhachidian vibrotherapy*."

For the mental depression, insomnia, and other nervous symptoms of hyperpiesia, I have in several patients tried, and by certain experts well tried, "suggestion" (p. 110), but so far in vain.

Venesection we may consider from two points of view—from that of prompt relief at a crisis, and, again, from that of a gradual remedial means, repeatedly and systematically used over long periods of time. A crude measure no doubt it is, but it may be serviceable until more scientific methods are available. By a single large bleeding a threatening condition of high arterial, or high venous, pressures may be averted for the time being; or a pulmonary œdema dissipated, or an eclampsia cut short; but can we by bleedings repeated at intervals mitigate or cure an obstinate hyperpiesia? On critical bleedings, as in pneumonia or mitral disease, I have not in this place to dwell; our concern now is with Hyperpiesia. If we have reason to believe that a persistently high blood pressure is associated with chronic renal disease, a disease apt to impoverish the blood, at least in respect of its red corpuscles, we may hesitate to bleed so freely or so frequently as to be of much use in reducing high pressures; but if the state be one of hyperpiesia, a disease in which usually the blood is not thus reduced in value, and may indeed be hyperglobulous, is venesection here likely to be at least harmless, or has it indeed proved to have a curative or moderative effect? At first sight, it is true, the promise would not seem great. If we reduce the content of the peripheral vessels, and they contract down upon the smaller content, unless the supply of blood is to fall, the velocity must be increased; that is, the heart must work so much the harder, and for the time we are back where we were. Furthermore, in a few hours, or in a day or two, as resorption proceeds, the vascular system will refill. For this however the vessels may dilate—it appears that they do—and thus pressures may not rise, though, as there may be a reduction in blood value, a larger quantity of blood may have to be delivered in unit of time. Cook and Briggs[1] say that experimental venesection on the one hand, or transfusion on the other, cause on the

[1] Cook and Briggs, *Johns Hopkins Reports*, 1904.

pressure curve but transient drops and rises respectively—acute angular excursions. Of these phases all old pupils of Ludwig and Roy were well aware; but such experiments do not tell us of the subsequent effect on plethoric men. Saline, it is true, is readily absorbed, and transfusion of blood is efficient in raising and sustaining arterial pressure, yet the effect is transient. If we turn to experiments on the normal animal, we observe that a rise of venous pressure does not, within limits, raise arterial pressure, either generally or locally; but in mitral regurgitation, where venous pressures had gradually risen, Dr. Mann [1] observed that bleedings of 10-23 oz. (in adults), by abating irregular maximum readings, and enabling the diastolic to pull up, steadied the heart. At first there was no gain in mean systolic pressure, but in about four hours it would rise, and in all his cases did so. Again we are told that on venesection there is an increase of agglutinins, and probably of other "antibodies." Evidently, here again we have very complex conditions to deal with, and clinical experience must not yet be disarmed by experimental bleedings on normal animals; nor indeed by the fluctuations of single symptoms of the series. Besides, as we have seen (Vol. 1. p. 86), we are ill-informed on diastolic pressures, with which nevertheless in this problem of venesection we are very closely concerned. My impression is that venesection has some abiding effect of moderation of diastolic pressures.

Let us turn then to the evidence of empirical treatment. Of the vast abuses of bleeding I will say nothing; I remember the last of them too well, in Bouillaud's wards. As in Sangrado's day in Western Europe, so of this fashion in ancient Rome Celsus sagely writes: "Venesection is no new thing; what is new is that there is scarcely any malady in which it is not practised." But amid the crowd of Sangrados there were, as amid all controversies, wise and thoughtful men who used their remedies with sagacity and economy; such men, for example, as Wepfer, of whose sensible practice we may read in his book on *Apoplexy*. They bled "not pale and delicate persons, but those who loved meat, generous wines and choice dishes . . . such have much blood, their urine is thick, and some ebullition of humours is

[1] Mann, *Guy's Hospital Gazette*, Jan. 25, 1908, and *Guy's Reports*, vol. lxii.

to be feared in them." Now such urine indicates not chronic renal disease but hyperpiesia. Lancisi, after pointing out the benefit of blood-letting in plethora, proceeds to warn the reader against bleeding too heavily, "which may lead to cachexia and dropsy." He reflects "adeo verum est prudentiam in medico potiorem esse virtutum partem."

When we leave the ancients for our own day, we may ask ourselves if now we have not gone, in this respect, to the opposite extreme of inaction. We are indisposed to listen to the testimony of our fathers on behalf of bleeding; partly for the good reason that they had not modern methods, nor modern thoughts moulded on modern methods, wherewith to criticise their own results. Yet it is not improbable that, in their confused experience of bleeding, our fathers were encouraged in their practice of it by a substantial, if indiscriminate, admixture of good results with bad. However, more absolute, and perhaps no less indiscriminating, has been the revolt from venesection, so that it is not easy now to obtain evidence concerning this practice, for good or harm. In my own practice I have found experience very hard to attain. Only in a small minority of cases otherwise appropriate for this means are the patients, or their friends, or their family physician, ready to agree to periodical bleedings; and of those in whom it may have been advised and practised, too many pass unrecorded out of sight and memory.

Yet of venesection for Hyperpiesia I have some increasing knowledge. I began some years ago to use it where I dare, and I had experience enough to recommend it, for Hyperpiesia, in 1907.[1] Recently I have had under observation for some time three patients who have been bled at least twice a year. They look forward hopefully to the operation, declaring that the symptoms of excessive blood pressures are marvellously relieved by it. And a permanent improvement seems to be thus maintained; but these three cases are beyond the stage of cure, though not of substantial amelioration. It was in such cases, which they recognised by the tight pulse, that our forefathers were wont to take blood freely; and, I believe, with great benefit. One lady, who has now been bled for some years, after the first bleeding wrote to me a letter of the warmest grati-

[1] C. A., in *The Hospital*, Nov. 17, 1907.

tude, saying she had found astonishing relief from it, and that many obscure and troublesome symptoms had been dissipated. Her own medical man—Mr. Day, of Baldock—added his testimony to the same effect, and told me that he and his patient together had learned with some precision when venesection should be repeated. Swollen veins on the arms and hands are among the indications of the need. Usually the interval is about six months; but if the operation seems to be required sooner, there need be no hesitation in shortening this interval. This lady, in later middle life, had been subject for ten years or more to very high and sustained pressures, with a tightly contracted radial artery, and an enlarged left ventricle. There have been no renal symptoms or signs. I cannot doubt that venesection has not only ameliorated her discomforts, but has greatly prolonged her life; for the range of her blood pressures has been very materially moderated from about 200 to about 160.

As regards the testimony of others, I may refer to Felix Mendel of Essen,[1] who, while disclaiming "Vampyrismus," illustrated some judicious remarks on venesection by cases of which I will quote one example, that of a male, æt. 50.

There was a very high range of sustained arterial pressures. Family and previous personal history good. No infection. Complained of vertigo and distressing fulness of head and headaches. Pulse very tense. Second aortic sound very loud, and left ventricle hypertrophied. No albumin in urine, or other renal sign. No sugar. Was bled (200 cm.) with great relief, especially to the head; and under vegetarian diet with milk and massage, and iodide of potassium for three months, was better every way for eighteen months. Then returning symptoms indicated a second bleeding, which was likewise efficacious. After a while the symptoms increased again, but for some reason reliance was placed on diet, he was not bled; he grew worse and fell in an apoplectic attack with hemiplegia.

Mendel thinks that for a long time after venesection the viscosity of the blood is less, the stream rate accelerated, and the heart's work reduced. Although Mendel is the more inclined to bleed if the carotids are full and throbbing, and the face ruddy, yet he recognises also the many high-pressure cases in which, in

[1] Mendel, F., *Ther. d. Gegenwart*, July 1907.

spite of a spare frame and sallow countenance, it is none the less needed. A more rapidly oblique descent on the sphygmographic curve on compression of the brachial suggests a gain in velocity.[1] The apparent gain in velocity persists for a few days, though the systolic pressure may increase, but not to its former degree. Some years ago Dr. Oliver Withers, of Sidmouth, published in the *Practitioner* two cases of excessively high blood pressure with oppression of the head and vertigo, in which bleeding gave remarkable and persistent relief. Purgatives had availed little. Dr. Fortescue Fox[2] has had many opportunities of bleeding in these cases; a lady, suffering from high blood pressure, he bled regularly from the age of 55 to 60; the heart was large and labouring, and the peripheral vessels thickened; he attended this patient for twenty years for rising pressures which first showed themselves at the age of 48, but after the menopause became aggravated. Among other symptoms were fulness of the head, dyspnea, vertigo, and a slight apoplectic attack. As all ordinary means had failed to give relief, she was bled. The relief was striking and immediate, and for some time persistent. It was found necessary however to bleed her every six months for six years, taking usually from 10 to 20 oz. (on one occasion 24 oz.). At length something like recovery was attained, for at the age of 68 she was reported to be in good health. He describes other like cases: in one of them, with threatenings of apoplexy, an immediate bleeding, and two in the year following, gave permanent relief. As I write these words he is using this method successfully in the case of a gentleman of middle age whom I had seen with Mr. Wingate. Eulenmeyer[3] narrates many instances of repeated bleedings in these hyperpietic cases. By it, he says, viscosity is reduced (p. 147), and velocity promoted. He prefers to make small and frequent venesections. Broadbent practised venesection not infrequently,[4] and urged its use in high-pressure cases, especially in patients menaced by apoplexy. If the immediate fall of pressure after venesection be not considerable, 30-40 hours later it becomes

[1] See also Lewis, T., *Practitioner*, Feb. 1907.
[2] Fox, F., *Lancet*, April 23, 1910, and Nov. 4, 1911.
[3] Eulenmeyer, *Deutsche med. Wochenschr.*, 1906.
[4] See Broadbent, Sir W., *Treatise on the Pulse*, 1887; *vide, e.g.*, p. 287.

more definite, and the pulsations more ample; as if the bleeding had relaxed arterial spasm. Ortner testifies to the same effect.

In hyperpiesia venesection is not only well tolerated—even 40-45 oz. may be taken before the radial pulse softens—but its effects seem far-reaching. It compasses some profounder alteration lying beyond our direct appreciation, emancipates the system from some burdens unknown; cuts vicious circles perhaps, or alters the solids of the body. Under no head of treatment is the discrimination between hyperpiesia and decrescent arteriosclerosis more imperative. This remedy, so efficacious in hyperpiesia, in the decrescent form of sclerosis would rarely, if ever, be called for, and might be disastrous. And yet influential physicians are now talking of "bleeding in arteriosclerosis"!

Dr. Herbert French[1] has proposed, as a less formidable operation, "venepuncture" instead of venesection, the blood being drawn off through a hollow needle. He thinks this method might facilitate small and frequent bleedings, if these be preferred.

When headache is severe, the brain oppressed, and the blood pressures very high, *lumbar puncture* may be thought of. I have never used it deliberately for a hyperpietic case, but I have used it for like conditions in chronic renal disease with advantage. I think I got the suggestion from Vaquez; the operation is one which might come more into use in phases of these maladies.

For those physicians who seem to know that high pressures are "compensatory," and therefore to be maintained, most of our therapeutical means will seem worse than idle. I cannot pretend to be more than an empiric in this matter. Yet it is with some disinclination that now I pass from the physical methods[2] to the *use of drugs*. It would be ungrateful to forget that in hyperpiesis we owe much to certain drugs, but it is reasonable to remember that in adding drugs to toxins we may be piling poison upon poison without full recompense.

[1] French, H., *Guy's Hospital Gazette*, Feb. 15, 1913.

[2] The Greek physicians of the Empire (Celsus, Galen, Antyllus) called bleeding, cupping, purging, emetics, massage, gymnastics, fasts, sweats, their "common aids" (*κοινὰ βοηθήματα*, or *προσφερόμενα*).

In entering upon the discussion of direct *vasodilators*, as contrasted with agents which bring about this change indirectly by modifying the causes of morbid constriction, we have to consider how far mere dilatation—brought about, that is to say, immediately and singly—serves any good purpose. We are told that to act thus directly upon the vessels is but to "treat a symptom," and is therefore absurd. But whatsoever be our judgment on this or any such particular effect, the common denunciation of "treating symptoms," which sounds very philosophical, is surely but a parrot phrase. Why should we not treat a symptom? If in Granular kidney by mere pressure reduction the grievous headache be abated, or if in angina pectoris the pain be thus charmed away, we have so far at any rate a substantial gain. In renal disease it is generally agreed that on the whole, with due caution, to lower pressures is helpful. Moreover, if by mitigation of his suffering the patient gets a chance of picking up in many other ways, are we not more than justified in our interference, narrow as it may seem? We never know what interference may cut a link in a "vicious circle."[1] To deprecate a therapeutical means as mere treatment of a symptom is idle, unless by this step we are doing some incidental mischief. If we cannot stop the crack in the water-pipe we need not throw away the mop. The warning should run not against the treatment of a "mere symptom," but lest while giving our attention to the symptom, and snatching at an immediate advantage, we lose our grip of the case as a whole. If it be argued that, in chronic nephritis for instance, the high pressure is a cardinal condition of an artificial but critical balance, and that to reduce it without counteraction of its causes is to upset the unstable machine, we shall reply that the appeal must be to clinical experience, to empirical results. We have gone back to learn of nature, though not obsequiously. That ultimately we shall have to be guided by the intimate physiology of these processes is no doubt true, but true of a more or less remote future; at present we are working in the dark, and must grope by touch. It is too readily assumed, without any definite data, that every natural coefficient is for the advantage of the system. How do we know

[1] See Dr. Hurry's book on *Vicious Circles in Disease*. "One could win the fight with the other fellow if his terrier were not biting one's leg."

that, in current jargon, these high pressures are "compensatory"? How do we know that they are not the result of some poison acting upon the bulb or other vasomotor centres, driving up the pressures in sheer mischief, and straining the mechanism to no purpose? We do not know even the outlines of an answer, and seem not to be near such knowledge. We are agreed that when called to a patient whose machinery has thus for some time been carried on upon a compromise, if we cut in upon an abnormal but provisional balance, if we depress one pan of it too sharply, we may endanger the temporary adaptations, and even add to, rather than alleviate, the patient's discomfort; but if brusque practice of this kind may be unwise, a more circumspect intervention in the same direction may be as wise. Indeed under urgent circumstances, such as stenocardia, or menace of apoplexy, a brusque interference, with vasodilators or otherwise, even at the cost of some other obliquity, may be imperative; and, with time before us, we may continue such remedies with caution and discernment, in combination with other measures which we flatter ourselves are more rational and comprehensive.

Whether then as swords for a knot, or as more continuous aids in perplexity, whether quickly to lull the agony of an angina or slowly to moderate the stresses of an arterial plethora, the *nitrites* are often valuable, and sometimes invaluable means; but we must use them with this discernment. Their effect in lowering arterial pressure, it may be by 25-30 mm.,[1] is prompt and remarkable; and in hyperpiesis lasts longer than in health. When pressures are acute and menacing, we shall betake ourselves to the Liquor trinitrini; for more continuous use sodium nitrite (usually prescribed in solution with 5 or 10 grains of potassium nitrate) is more suitable, and usually answers well. Unfortunately however the drug is unstable, and apothecaries' samples are variable. It varies least in tablets, yet of one sample an ordinary dose may be almost too prompt in its effects, whilst of another a dose three times as large may appear inert. Not only so, but the margin between therapeutic and toxic doses is somewhat uncertain, and vertigo and nausea may ensue on a dose too large for the individual (Vaquez).

[1] See Matthew, E., *Quart. Journ. Med.*, April 1909. Dr. Matthew reports comparative tests of several of these agents.

The degree of tolerance of nitrites varies considerably in various patients; the first doses should be prescribed with caution. This is especially true of sodium nitrite. Some persons bear all nitrites badly, when not doses but frequency of doses may be increased. This salt must not be combined with potassium iodide, nor with any oxydising agent. Erythrol tetranitrate may be better suited for prolonged depressor influence; being less soluble its effect comes on more gradually, and is more persistent than that of sodium nitrite, as this in its turn is more gradual and persistent than nitroglycerine. One of our medical graduates—Dr. Newmarch—in a private letter told me he had found long courses of erythrol efficacious in some melancholic patients with high blood pressures. This drug is said, by a gradual moderating influence, to restore sleep to those sufferers from high pressures in whom insomnia is a distressing symptom.[1] As a chronic remedy then erythrol is convenient, but it is explosive and, like sodium nitrite, unstable; still if kept in a cool dark place it should last well enough for a season. It is best given in tablets of a half to one grain, or more. But I shall be pressed to say plainly if I advise the use of vasodilators systematically and continuously in all high-pressure cases. To this question I am almost ashamed to confess that I cannot give a definite answer. I think not. When the heart is beginning to yield nitrites must be avoided, or, if urgently needed, guarded with digitalis. Rest in bed would be essential, and the doses anxiously watched. If there be reason to infer loss of conductivity, even this combination may be unsuitable. But we cannot enter here upon the field of cardiac therapeutics. My own practice is to try first what diet, frequent calomel (in non-renal cases), and saline laxatives will do; then, if more interference be needed, I try courses of physical methods, of baths, of the high-frequency current if available, and so forth, and sometimes venesection; if, after a fair trial of these measures, some relaxant seems called for, I then betake myself to a systematic if not prolonged course of vasodilators, watching their effect on all sides; yet always under the prejudice that there is something too artificial, too rule-of-thumby, about the method.

[1] Bruce, M., *Sc. Med. and Surg. Journ.*, 1900.

How is the depressor effect obtained? It is agreed that diastolic pressures are lowered. Is it by peripheral dilatation, or wholly or in part by splanchnic dilatation? If by splanchnic dilatation only, what do we gain? Apparently nothing more than a transference of part of the bulk of the blood from one area to another. And may not the heart then endeavour to counteract this kind of fall by a quicker rate, more energy, and more output per minute, so as to restore the systemic arterial pressures to their former levels?[1] Again, one cannot feel sure that the depressor effects of the nitrites are merely by vasodilatation; I ponder uneasily whether some part of the relaxation be due to a lowering of the cardiac energy, with a consequent fall of stream velocity. It is stated indeed by Russell-Opitz[2] that nitrites accelerate the stream in the femoral artery, and by others that they tone up the heart; but we cannot argue from the normal animal to the abnormal man—or even to the normal man, whose splanchnic mechanism is correlative with the upright attitude. Certainly during their use the heart sometimes dilates, and there are phases in which hypotensive medication seems mischievous. Unfortunately, the consultant can watch few of his cases so continuously as to enable him to form any steadfast opinion about long courses of treatment. In angina pectoris, it is true, one sees nitrites used in large doses continuously for many months, and from these perseverances I have seen no harm; but here the arterial pressures may not be deranged. For hyperpiesia, then, I cannot at present advise the use of nitrites as more than adjuvant, when some rapid relief is called for, as in stenocardia or paroxysmal dyspnea. The doses ordinarily prescribed are but initial; to produce a well-marked effect, sodium nitrite may be pushed carefully up to a rate of eight to ten grains, or more, in twenty-four hours—with twice as much saltpetre—but in frequent and limited, not in larger and rarer, doses. Some physicians indeed, relying upon an

[1] Since these words were written I have come across a paper by Laidlaw (*Journ. of Physiol.* vol. xl., 1910), who, experimenting with a depressor drug (tetrahydropaperveroline, not as yet suitable for medical use), recorded just this effect: the dilatation was chiefly abdominal (splanchnic, etc.), and the cardiac rate, and output, and energy of beat, were so much increased that (under normal cardiac conditions) the arterial pressure did not fall much, or for long. The circulation time must have been shortened.

[2] Russell-Opitz, *Journ. Exp. Med.*, 1910.

acquired tolerance of nitrites (Osler, Pässler, Vaquez), run them up to very large doses, to m. 30-40 of the liquor trinitrini, for example, without apparent harm. In all these therapeutical tests we should try to watch not systolic pressures only but also the minimum, and therewith the amplitude. A run down of diastolic pressures, like a rise of rate, is ominous. I have never seen a "nitrite habit" established in hyperpiesia, but I have seen it in angina; a malady in which the patient's sufferings may justify his addiction to the remedy.

Dr. Hobhouse kindly reported to me the following observations made by a medical man upon himself while at rest. He was the subject of aortic aneurysm, and I had seen him in consultation. He used tetranitrin in slightly increasing doses. His pressure on the first day, at 11.30, was 136; at 2 o'clock it was 128; at 5 it was 137; *i.e.* it returned to the former and usual level. But at 8.30 (without another dose) it fell to 120. Another dose was then taken, and the pressure fell to 112. This experiment was repeated day by day, falls of 136-120 or thereabouts being noted. The fall usually became apparent in five to thirty minutes, and occupied about five hours, when it would rise again to its previous level. But the observer found that without the drug the fluctuations were nearly as large. In spite of cutting down food, alcohol, or tobacco, rises would occur; and the tetranitrin did not always cause a fall. After lunch, whether it were with or without a little alcohol, there was always a rise. On one day a pressure of 140 at 8 A.M. fell, under the drug, by 10.30 A.M. to 123, but in seven hours more it had risen to 134. (See daily phases, Vol. I. p. 21.)

Vasodilator drugs then, when the pressures are acute or urgent, have a certain but not a predominant part to play. Of stovaine, alleged to be a vasodilator, I have no experience, nor have I any important information concerning it in this respect from others; nor again of sparteine, proposed as a vasodilator remedy by Professor Dixon. Acetylcholine is said by Reid-Hunt[1] to have an amazing depressor activity, "a hundred times more activity" in this sense than has adrenalin in pressor activity, and a hundred times more activity in the depressor sense than nitroglycerine; but its mode of action and its possibilities in thera-

[1] Reid-Hunt, Sect. Physiol., Brit. Med. Assoc., Toronto, 1906.

peutics are unknown. Choline we know, by Dr. Mott's experiments, to be a depressor by way of the heart, not a promising function for therapeutical values. Both choline and acetylcholine are opposed by atropine, which suggests that those substances act through the vagus; but it is not easy to see how by this route acetylcholine can exert effects so enormous. By thyroid extract, given in ordinary doses in high-pressure cases, I have gained no advantage; but the doses of 30 to 40 grains of which we read assuredly ought to set up some disturbance, good or bad. Dr. Oliphant Nicholson,[1] speaking of high pressures in pregnancy, says that in this condition thyroid extract is very efficacious; headache and other untoward symptoms are abated, and the transition to well-being is remarkable.

Of the more specific drugs in hyperpiesia, none is equal to *mercury*, especially—for the many who tolerate it well—as calomel; for others, blue pill. The calomel is to be given in fractional doses, say $\frac{1}{4}$ grain once, twice, or thrice daily for four or five days; or $\frac{1}{2}$ grain every night: the course to be repeated from time to time. A saline laxative is required by many persons; in others the calomel may have to be guarded by a grain or two of Dover's powder. The patient soon learns when to repeat his course, and to expect the benefit to be had from it. In the intervals, small frequent doses of podophyllin—$\frac{1}{10}$ to $\frac{1}{4}$ grain thrice daily—or of euonymin or iridin, may be prescribed. From day to day some notion of the effects may be guessed at by the finger, but the blood pressure should be measured at least once a week. It is desirable, as I have said, to keep the patient in ignorance of the figures, lest he worry about them, or disturb the record by expectant attention. In the cases of intercurrent high pressures in elderly persons, with or without decrescent arteriosclerosis, mercury is marvellously efficacious; by its means insomnia and other discomforts are often dispelled at once. Of other laxatives some are useful, not so much for expulsion of water, as to provide for excretion, and for the activity of the lymphatic system. Thus the morning saline draught, though by itself it is not efficacious, is a valuable aid to other measures. In several instances of hyperpiesia I have been surprised to find how little amendment long courses

[1] Nicholson, O., *Brit. Med. Journ.*, Oct. 3, 1903.

of laxative saline achieved alone; and this in cases which afterwards responded to calomel. They may increase viscosity. A good habitual laxative is the old-fashioned electuary of sulphur, senna, pepper, tamarind, etc.

The general faith in the *iodides* as moderators of high arterial pressures is curiously robust and perennial, notwithstanding the default of any definite evidence of this virtue in them. Their reputation was first gained in the field of aneurysm, a somewhat dubious test; for, as about 90 per cent of aneurysms are syphilitic, the virtues of the iodides in syphilis may bring about some amelioration by way of their specific influence. It was by Sée and Lapique [1] that they were carried forward into the general field of arteriosclerosis. Pal seems to me to speak in exaggerated terms of their value in reducing high pressures. Syphilis apart, I have never been able to satisfy myself that the iodides, either as vasodilators, by reduction of viscosity, or otherwise, have any effect, direct or indirect, in abating arterial pressures (see Viscosity, Vol. I. p. 145). Still, we have to meet a heavy phalanx of empirical observers on the other side. Dr. Edwin Matthew (*loc. cit.*) classes the iodides with the nitrites as pure vasodilators, especially in hyperpiesia before vascular strain has appeared; a remarkable definition, for Huchard, as definitely, deprecated their use in the "presclerotic" period, the only period in which Matthew, and Graham Steell with him, thinks they are of service. Matthew begins with 10 grains thrice daily, increases the doses rapidly for some days, and then decreases them. Ludwig Braun of Vienna follows the same method.[2] In my own practice I have not followed this method precisely. Ortner's method [3] is different from that of Dr. Matthew. He orders 3 to 5 grains of potassium iodide, with a little sodium of carbonate, copiously diluted, thrice daily after meals; omitting each week a day or two, but not more. Acids in the meal should be avoided shortly before the dose. He advises against its use in renal disease or in anæmic conditions; and it should not be given while the patient is under calomel. Von Romberg, Klemperer, and Ewald still prescribe the salt in moderate doses. Sir L. Brunton says

[1] Sée and Lapique, *Rev. de la science méd.*, 1876.
[2] Braun, L., *Med. Klinik*, No. 26, 1911.
[3] Ortner, *Jahreskr.*, Feb. 1911.

many patients can tolerate a dose of 25-30 grains at bedtime, well diluted, when 5 grains thrice daily cannot be borne. He also believes that the drug is effectual in abating pressure.[1]

Various explanations are given of the virtue of the iodides in alleviating excess of arterial pressure but, as in the old story of Charles II. and the bowl of fish, without first ascertaining whether the alleged effect follows, or not. In so far as therapeutics are to be rational, before we discuss the mode of action of a remedy, we must clearly understand the problem before us. Some authors assert that these salts act thus by releasing arterial spasm (Ortner, Matthew); some that they act by reducing viscosity (Erlenmeyer, Romberg, Ludwig Braun of Vienna, Martinet and others); others, by the oracular assumption of "some kind of metabolic resolution or elimination." Müller and Inada's paper,[2] in which these investigators alleged that potassium iodide reduces arterial pressure, not directly but by reducing viscosity, is discussed in the section on Viscosity (Vol. I. p. 145). Their statements found much acceptance in Germany; but we have seen that, although viscosity must be a considerable factor in the labour of the circulation, upon arterial pressures it has less effect than was anticipated; though notwithstanding the cardio-arterial work may be much affected. However, it was upon this paper that Romberg's and other opinions [3] on the influence of the iodides were based. Dr. H. D. Rolleston suggests that the abating effect may be compassed by a mediate excitement of the thyroid (p. 134). Erlenmeyer, on his hypothesis, orders the iodides to be administered in long courses after the manner of Ortner, and combines this treatment with small and frequently repeated venesections.[4] Erlenmeyer's article was written to dispute Krehl's objection to their use, which was not that they lack the effect attributed to them, but that they were too effectual in reducing the high pressures, which in "arteriosclerosis" Krehl believes to be essential to the defence. Krehl is beset by the notion that any reaction in a perverted body is compensatory, and so to be respected. Hochhaus of

[1] Brunton, Sir L., *Clin. Journ.*, August 28, 1912, sect. vii.

[2] Müller and Inada, *Deutsche med. Wochenschr.*, 1904, No. 48.

[3] Romberg, *Deutsche med. Wochenschr.*, Aug. 31, 1905, and in his book (*Herzkrankheiten*, 1906).

[4] Erlenmeyer, *Deutsche med. Wochenschr.*, 1906, No. 7.

Cologne,[1] a very able physician, urges long courses of iodides, but prescribes also diuretin, the milder baths, and exercises. But he does not make his argument clear, he regards arteriosclerosis throughout as one disease, not distinguishing between its several modes and issues. In this confusion treatment goes utterly astray—perhaps with all of us.

However, in the present debate the argument may be about moonshine. We have had it on the high authority of Stockman and Charteris [2] that in man the iodides, pressed even up to iodism, fail to bring about any fall of blood pressure, whether by dilating the arterioles, by reducing the heart, or otherwise. In animals they injected sodium iodide intravenously (the sodium salt to avoid any effect of potassium on the heart) without any pressure reduction, though as regards the heart Professor Stockman says that the amount of potassium in its iodide is so insignificant that it is scarcely worth while to substitute the sodium for the potassium salt. Dr. James Burnet,[3] by the test of subcutaneous injections of iodipin, came to the same negative result in man. It is sometimes forgotten however, especially in experiments on animals, that in certain morbid states reagents may prove efficacious without manifesting the like phenomena under the conditions of health. My attitude for the present is to remember that many experienced observers are convinced that in high arterial pressures the iodides are potent for relief, and that accordingly, unless we ourselves have some strong reason to the contrary, we are in a measure bound to give our patients the benefit of the chance. Josué likewise seems to continue to prescribe them, although he frankly says that, however administered, he has never seen any benefit from them. The difficulty is that to many persons iodine is always a nasty and often a depressing drug, and yet one which, if it is to be of any use, must be given in full doses and over a long period. My custom therefore is to explain the position to the patient, and to invite him to try the drug at will; some persons have less distaste for it than others. I have tried many of the preparations alleged to be less disagreeable than the ordinary salts of

[1] Hochhaus, *Deutsche med. Wochenschr.* vol. xxxviii., 1912.
[2] Stockman and Charteris, *Brit. Med. Journ.*, Nov. 23, 1901.
[3] Burnet, J., *Lancet*, Sept. 6, 1906.

iodine, but without any notable advantage. Some physicians of repute recommend, especially for cerebral symptoms—headache, vertigo, loss of memory, or speech arrests, and paræsthesiæ—"tiodina" (whatever this may be) in subcutaneous injections of 3 grains, for 16 doses. They are said to be painless. For "arteriosclerotic" (high pressure ?) headache Neusser prescribes a combination of bromide and iodide. It will be seen that from any point of view these clinical records, as described, are too often a muddle of high pressure and decrescent disease.

If a case be altogether or in part syphilitic the iodides are of course indispensable; but in the arteriosclerosis of syphilis, as such, local or general, the blood pressures are not raised. In this infection the relief given by these salts is often marvellous, especially to the cerebral symptoms; but no such case should escape a full course of mercurial treatment. In arterial disease of syphilitic origin, unless the heart be much degenerated, or the lungs emphysematous,[1] salvarsan also should be used carefully but promptly by an expert.

It is impossible here to enter fully upon the treatment of syphilis. How successful specific treatment may be, and permanently successful, in curing cerebral and other arterial disease of this kind is happily well known to us all. As a mere illustration I insert the following case:

At the Leeds Med.-Chir. Soc., March 20, 1903, Dr. Churton showed a woman, aged 39, who had been under antisyphilitic treatment since December 1901. At first the subclavian arteries, especially the left, were thickened and greatly dilated, the pulsation being visible above the inner and below the outer part of the clavicle (see Vol. I. p. 395). The carotids were similarly, but less, affected, and pulsation was felt behind the manubrium. All these arteries now appeared perfectly normal, and the other symptoms were relieved. Of course the speaker did not assume that the case could be dismissed as cured. We know but too well how relapses will occur. It is to be hoped that salvarsan may control them.

To judge by the mode of action attributed to the iodides, these salts would probably not be recommended for decrescent arteriosclerosis, in which state arterial pressures are not char-

[1] See Grassmann, *Münch. med. Wochenschr.* No. 42, 1910; a helpful article.

acteristically augmented; if perchance they rise episodically (Vol. I. p. 452), mercurial and other remedies for this transitory condition are more efficacious. However, I am driven to repeat that in the published papers the confusion between the terms arteriosclerosis and high arterial pressures, or the acceptance of arteriosclerosis as "a disease," is so incessant as to make their message too often unintelligible. Erlenmeyer (*loc. cit.*) indeed looks upon arteriosclerosis without high pressures as so eccentric and improper that he advises a cautious raising of the level by gaseous baths at temperatures about 30° C.

For raising pressures, in case of a lurch to the falling side (usually a heart failure), Dr. H. C. Mann (*loc. cit.*) found no drug equal to strychnine. He says that morphia also often acts well in raising diastolic pressure and steadying the systolic. He finds also that in "second wind" the diastolic pressures rise and the systolic abate in the same way.

Of other salts in the category of remedies there is not much to say: nitrate of potassium is an old diuretic, and in hyperpiesia is conveniently prescribed with other and more specific agents; it often contains some nitrite. Salts containing much potassium may reduce pressures, but at the expense of cardiac energy, the very opposite is what is needed. Dr. Gee used to say that whey—a breakfast-cupful two or three times a day—has an excellent effect in dissipating uratic deposits. Ammonium chloride is said to have a relaxing effect on the vessels, while leaving the heart intact; I have no experience of its value—it may merit more attention than it has received. The part of sodium chloride, the alleged retention in high pressure of sodium chloride in the tissues, and its comparative scantiness in the blood, the urine, and certain viscera I have discussed already (p. 93). Certain authors recommend tablets of sodium chloride, sodium sulphate and phosphate, magnesium phosphate, and calcium glycerophosphate in due proportions, for "arteriosclerosis"; it is not easy to say why. Of the calcium salts we shall hear more presently. Dr. George Oliver at one time recommended the hippurates as moderators of high pressures—he knew better than to speak vaguely of remedies for "arteriosclerosis"; but subsequently I think he preferred to them the benzoates of potassium. A diuretic influence should be the sign

of their benefit. Gouget recommends sodium silicate;[1] he gives 1-3 grams of a 35 per cent syrup much diluted, and reports fall of pressure in "arteriosclerosis" (presumably he means hyperpiesis?), and therewith relief of vertigo, headache, etc. He states that by a previous administration of sodium silicate the damage to the rabbit's aorta by adrenalin may be prevented.

In respect to the salts of calcium there is not much to be said of therapeutical value. In passing from practice under the conditions of the extremely soft waters of the West Riding to the chalk and gypsum waters of Cambridge, I have not observed any difference in the incidence or mode of arteriosclerosis or blood pressure. That without excessive pressures lime salts are conspicuous in arterial decay we know well enough, but we know also that this deposition of the lime is but a secondary event. If an excess of lime in the blood augments the viscidity of this fluid we shall be careful to avoid it; but we are far from assurance that such is the case. Opinions differ widely on this matter; our own investigators, such as Professor Dixon and Sir James Barr, and many foreign observers likewise, hold sundry opinions.[2] Certainly in the malady before us we are no gainers by administration of lime salts, and in this obscurity we may as well forbear from any use of them in medicine or diet; but let us be clear at any rate about the maladies with which we are dealing; let us not continue the habitual confusion of distinct affections under the name of "arteriosclerosis"; let us recognise that if lime salts be early and conspicuously deposited in decrescent arteriosclerosis, in which blood pressures are not much affected, in hyperpiesia, in which augmented pressures are the first obvious deviation from the normal, lime deposits are tardy and, at any rate till protracted stages, inconspicuous. Thus the effects of lime ingestion, if any, on the regulation of function, and on the coats of the vessels, respectively must be considered apart; the processes are independent. The heart cannot be short of its own calcium requirement.

[1] Gouget, *Presse méd.*, Dec. 6, 1911, quoted *Arch. des mal. du cœur*, avril 1912.

[2] See *Report*, Brit. Med. Assoc. Mtg., 1912. Rumpf was the author of the limeless cure. Voorhoeve, who has made a recent study of the subject, says (*Berl. klin. Wochenschr.* Nr. 36, 1912) (see vol. i. p. 499) it is possible to increase the lime content of the blood by administering it in large doses, but that there is no evidence that it has then any important effect.

The *antisclerotic serums*, or *tablets*, invented by Truneczek, Natterer, and others for the treatment of "arteriosclerosis," without regard to its kind or to the initiatory processes of its manifestation, are pedantic; and to the best of my belief useless. On empirical grounds, they are falling into discredit. Some of them are proposed on the shallow hypothesis that arteriosclerosis is due to an abnormal saline constitution of the blood. After the manner of other proprietary remedies, they have been advertised without criticism.

If *diuretin* (5-7½ grains *ter die*) be of service in arteriosclerosis, it must be in the secondary form of it, secondary, that is, to abnormally high blood pressures; it does not counteract mechanical strain, but it may counteract, or eliminate, the causes of vascular strain. The assertion that it relaxes vascular spasm rests, so far as I am aware, on no verified data. Dr. Haynes, in our laboratories, showed that diuretin increases the flow from the coronary arteries; but we also know that such outflow depends less on pressure than on activity of cardiac metabolism (Barcroft and others). That it has some virtue in high pressures I believe, on empirical grounds, for in angina pectoris it seems to moderate relatively high arterial pressures. On its effect in hyperpiesia we need some careful continuous observations; somehow or other it seems to do good in this disease. It should be prescribed in as little water as possible. *Aspirin* and sodium salicylate are prescribed also in excessive blood pressure. In one case, which came under my notice, we quickly relieved a grievous cough and bronchitis, which had tormented the patient for five winters, by reducing the diet by one-half and administering aspirin; it was not easy to discern which of the two was the crucial step, but the amendment certainly was accompanied by a reduction of the arterial pressures which before had ruled too high. Twelve years later this patient died of angina pectoris; five years before this event the arterial pressures had again gone up, and sclerosis of the mitral valve had given rise to an audible murmur. Dr. Core (*loc. cit.*) thinks (without experiment?) that sodium salicylate "neutralises some pressor toxins." Guaiacum, for its supposed virtue as a hepatic stimulant, is credited with a "katabolic neutralisation of pressor excretory toxins"; it may be worth a trial, and a better explanation.

Adonis vernalis has been recommended—two grammes of the extract (better than the infusion) per diem [1]—in more than one quarter, for rising blood pressures, and even for "abdominal angina."

Viscum album, at one time classed with the digitalis group, more recently has been credited by certain French physicians with a depressor influence upon the vasomotor centres and arterial muscle; and also with a diuretic property. Ortner,[2] on the contrary, found the fluid extract quite useless. *Yohimbin*, which for a time had a run as a pressure reducer, seems also to be useless,[3] though Fränkel has some faith in it; Ortner also still recommends *vasotonin* (Lippmann), which is a combination of yohimbin and urethane. This remedy is supplied in sterile bulbs for subcutaneous injection, once a day for 20-30 doses; the pressure is said to fall with relief to the symptoms; but in Ortner's opinion the reduction, if harmless, is transient. He thinks diuretin and agurin more efficacious, but that even these are wanting in abiding effect. I suppose little more, if so much, is to be expected from *Senecio jacobœa*, which is described in small doses as an augmentor of arterial pressure, in large doses (0·8-1·0 gm.) as a depressor; fluctuations attributed to constriction or dilatation of the intestinal vessels. Aconitine, veratrine, and antimony prove at best to be useless (Cash and Dunstan). There is a little evidence however in favour of *arsenic*, which may have some adjuvant metabolic effect; it is said by Coley [4] to be not a direct reducer of pressures, but by more intimate alterations to bring about an improved condition in these cases. Ortner also has a good word for it in certain cases where, in alternation with more direct treatment, a general "tonic" may seem indicated. The use of fibrolysins seems a blind suggestion; if they have any influence on the vessels it might well be an injurious one. When the heart is suffering under excess of pressures the inhalation of *oxygen* has been advised, in order to counteract any increases of viscosity by cyanosis.[5]

Professor Osborne of Yale and others have recommended

[1] *E.g.* by two Russian physicians quoted *Sem. méd.*, No. 25, June 18, 1913.
[2] Ortner, *Jahreskr.*, Feb. 1911. See also Selig, A., *Med. Klinik*, 1912.
[3] Pongs, *Zeitschr. f. exp. Path. u. Ther.*, April 12, 1912.
[4] Coley of Philadelphia, *Brit. Med. Journ.*, 1906, No. 24.
[5] See Bence, Julius, *Deutsche med. Wochenschr.*, 1905.

thyroid extract in hyperpiesia; I had already tried it without success, but perhaps my doses were not sufficiently liberal. Strychnine, in hyperpiesia, would be suggested only at a time of crisis, as of failing heart; but, as on occasion such aid may be desired, I may call attention to certain observations of Cabot,[1] which suggest that this drug does not of itself drive pressure upwards. He says, "The average pressure in the 50 cases that received a daily dose of strychnine was no greater than in 18 cases without any drug." The records varied of course, but "the total result was negative."

For the cardiac or respiratory distress hypodermic morphia, as first advised by me in 1869, has found acceptance. If on repetition its influence wanes, Fränkel[2] advises injection of heroin hydrochloride (0·0006-0·01); there is no call for increasing doses, though, like morphia, it does induce a craving.

The reference to remedies for yielding heart opens out the way to discuss that sovran drug, without which no discussion on cardio-arterial disease is complete, *digitalis*. There is an opinion abroad, and not without good reason, that in cases of high arterial pressure digitalis is contra-indicated. If we are still to suppose that digitalis promptly constricts the arterioles in most areas of the system,[3] and if we suppose also that hyperpiesia consists in such a general constriction, to propose to use digitalis in such cases would seem to magnify the evil. But now we hear that this constrictive quality of the drug rests on opinions which may need some considerable modification;[4] be this as it may, no physician would propose to administer digitalis whilst against an abnormal peripheral resistance a big heart was working not unsuccessfully. But when at length the brave heart is worn down by the intolerable strain under which for many years it has been too often permitted to strive; when, in spite of alleviated peripheral resistance, it is yielding in its walls, and perhaps leaking at its valves, when in an unequal strife it is giving way, our treatment will fall into line with the general

[1] Cabot, *Bost. Med. and Surg. Journ.*, Oct. 1, 1904.
[2] Fränkel, *Berl. klin. Wochenschr.*, 1913, No. 17.
[3] See Bradford, J. R., *Clin. Journ.*, July 27, 1904.
[4] Among other papers see Price, F. W., Section of Pharm. and Ther. Brit. Med. Assoc., 1912 (*Journal of Assoc.*, Aug. 17 and Sept. 21, 1912). Probably a good deal depends on the relation of the dose to the individual, and to his state.

treatment of heart failure. At such a time, if cautiously used and with full knowledge of the conditions of the particular case, digitalis is for mitigation invaluable, although the time for cure, or even for any abiding amendment, be now past. It is absurd to say that digitalis is inadmissible—this would be to treat a name, not a patient; but, on the other hand, when to exhibit it is one of the most critical dilemmas in medical practice. The strife is killing the heart; the state is an unnatural one, and only by some compromise is any rally possible. Traube saw clearly, then against adverse opinion, that at such critical stages of the malady digitalis must be admitted, and in his own practice he proved its efficacy. Dr. Graham Steell, while warning us against the use of digitalis in the earlier stages of hyperpiesia, recognises its value in the later, when the heart may yield. Sahli says, paradox as it may be, that when the heart is overdone by prolonged excessive pressures, and is dilating, digitalis must be used. Dr. Mackenzie however wrote that "digitalis is rarely of much service in dilated heart secondary to arteriosclerosis." May I, with the greatest respect, demur that there is not such a heart; or, if there be, that it is in decrescent arteriosclerosis with coronary obsolescence, a condition which neither he nor we had in view. The heart and the sclerosis of which we are now speaking are, on the contrary, consequent upon high pressure; the heart by hard work is forced and dilated, it is succumbing in the main not to diseased vessels, nor to intrinsic default, but to peripheral resistance by unknown causes; probably by extensive vasoconstriction. In all stages of such a strife it is clear that our first duty is continually to lighten the burden by whatsoever means may still be practicable; but, while not forgetting this, the heart must occasionally be lifted over a crisis. When the cart sticks in the mire we first lighten the load, yet, notwithstanding, a touch of the whip may also be needed to bring out for the crisis what reserve may be latent in the weary horse. But this metaphor, like many another, is not quite parallel, for the use of digitalis in this emergency is not the dilemma it appears to be; the case is not one simply of pull devil pull baker; there is, as I have argued elsewhere,[1] something more than a mere reinforcement of intra-

[1] Musser and Kelly's *Practical Treatment.*

aortic pressure; by the action of digitalis we gain some redistribution of pressures, whereby particular stresses may be resolved or alleviated. The rise of intraventricular pressure, if any, may be but momentary and tidal.[1] We shall not however betake ourselves to the drug until the heart is yielding; until then we shall pursue the more rational method of trying to reduce the resistance. If I were to make a clinical maxim I should say—turn to digitalis when diastolic pressure is rising and amplitude therefore narrowing. If signs of "stenocardia" appear we should perhaps avoid even a relief thus purchased at the cost of aortic tension, and still endeavour to soothe the strife by less direct methods—by rest, venesection, mercurials, and so forth. But this is not the place to enter at length upon the treatment of heart failure. If there be any indications of pulmonary œdema, venesection may be specially indicated. If digitalis is to be used I suppose that we must hearken to Dr. Mackenzie and Professor Dixon,[2] who assure us that the galenical preparations of the plant are as efficient as the active principles, or the much advertised novel preparations. But for this opinion of my colleagues I should have allowed myself too much reliance upon certain striking results with Nativelle's granules, which I have used since their first introduction; results due, it may be, to the more careful management and attention which a less familiar remedy requires.

We must not be led by the sallowness of some hyperpietics to give them the feeding tonics required by white flabby people.

How near after all modern medicine lies to the principles of our fathers, witness this extract from the *Tractatus de infirmorum sanitate tuenda*, Londini, 1726, by George Cheyne, M.D.,[3] an Edinburgh student, who practised in London and Bath:

Quae autem his neglectis Corporis Plenitudo succrescit, Lentoris Humorum et Infarctionis Canaliculorum parens; hanc diuturnorum omnium Morborum praeproperae et infirmae senectae, Vitaeque medio in Decursu recisae Causam facilè agnoscas, si attendas Evacuationum maximè Usu illos sanari, haec praecaveri. Venae etiam Sectione, Cucurbitulis, Vesicatoriis, Fonticulis, Catharticis, Geneticis, Sudori-

[1] Josué has some useful reflections on this subject, *Soc. méd. de Paris* Nov. 8, 1912.

[2] Dixon, *Quart. Journ. of Med.*, Jan. 1912.

[3] Cheyne was a pupil of Pitcairn.

ficis annon manifesta sit Evacuatio et supra Modum ingestorum Absumptio. Sed et ipsius Abstinentiae Exercitationis, Medicamentorum item alterantium; etc., etc. . . . Quod enim, Venae Sectio, quod Cathartica, quod Hidrotica praestant, idem in diuturnis Morbis (celeres enim lenta Medicamina antevertunt) praestat per Quatriduum aut quinque Dies Caena abstinuisse, Carnes non attigisse, Vino caruisse.

Decrescent Arteriosclerosis.—For arteriosclerosis secondary to hyperpiesis we have seen that much can be done; not, it is true, for the sclerosis itself, but for that exorbitance of arterial pressures to which it owes its origin. Indeed, but for the high pressures the sclerosis is, comparatively speaking, of little importance. In this case our argument must be not of the "arteriosclerosis," which is so incessantly put forth as "a disease," but of the hyperpiesia, or the chronic renal disease, one of which is the main morbid process. This, or these, we treat, not the sclerosis. So also with the decrescent mode; here again we treat, or should treat, not the sclerosis but the morbid changes, toxic or other, to which it owes its origin. In this mode of arterial injury the degeneration often depends more directly upon the condition of the vessels themselves; essentially it is, or often is, their own default; in hyperpiesis this is not the case. The contrast is therefore not directly between two primary diseases of arteries, but between a mode which is not primarily a disease of arteries at all but some one or more morbid processes setting up internal stresses under which the vessels suffer, and a degenerative process seated primarily or chiefly in the walls themselves. Now if this change be, as it is commonly called, "senile," we are as little able to "treat" it as we are to treat grey hair, or arcus senilis. Although the results of decrescent sclerosis may be far worse than of these, yet in principle our medical disability remains much the same. If there be any mitigation of local anæmias, of waning energy and heat, due to silting up of arterial twigs, it is these which we must deal with; how to arrest the vascular depravation which lies at the bottom of these failures we cannot tell; at any rate I know nothing of such means. Sometimes, it is true, decrescent arteriosclerosis comes on precociously; in these cases the lesions of the vessels, as revealed without to the touch and inferred within from the waning of function, are so malign as to suggest virulence, and

we find ourselves suspecting some instant toxic cause. Arterial disease of such kinds may be transitory (Vol. I. p. 288). But a precocious atheroma is usually accompanied by other and less equivocal signs of early senility, local and general; and is progressive. In more benignant cases however, although the vessels within reach may be greatly deformed, often indeed much more tortuous, lax, and calcareous than in the hyperpietic form, the general health is so little affected and life, speaking within limits, so little shortened or dimmed, that we may suppose the changes to be mainly of the long vessels of the limbs only, and, if so, relatively unimportant (Vol. I. p. 482). Too often however the memory trips, the flow of ideas is tardy, the feet begin to slur, the facial expression to fade; and we can do nothing but watch and lend a hand here and there as it may be wanted. Decrescent arteriosclerosis is immedicable. We "cannot hold time by the wings," and with some men it flies faster than with others.

In decrescent arteriosclerosis it is evident that we must not interfere with the moderate rise of pressure which belongs to all elder life; with increase of friction, and loss of the energy which the less resilient arteries now fail to restore, the stream velocity slackens; and if to meet this there may be some increase of cardiac energy—in some cases obviously there is, in others apparently not—we shall not try to counteract it. But we may try to improve the quality of the circulating blood, so that what reaches the periphery may be the better worth having. By airs fresh but mild and equable, we shall try to lessen the viscosity due to degrees of cyanosis, and to promote respiration in lung and tissue; by iron we may redden the corpuscles, and by gentle alteratives, gentle baths, and exercise carefully moderated within the nearer approaches of fatigue, we may try to keep the excretory organs at work. Concerning the conditions of the kidneys in this mode of arteriosclerosis, I have said enough in the proper section on this subject. On the other hand we have seen that in the decrescent disease an access of hyperpiesia may be attended with great peril to vessels already fragile. For these episodes the treatment recommended for hyperpiesia must be employed, and attacks of hyperpiesis, from which these patients are no more immune than other people, must be warded off;

happily in decrescent cases intercurrent plethora is even more readily dissipated than it is in early hyperpiesis.

Iodide of potassium, useful or not in hyperpietic cases, can be of little service in the senile. And the consequences are not those of the hyperpietic form. We have not to guard against apoplexy in its sanguineous form.

The heart, being more disposed to deteriorative than to hypertrophic change, is less apt to burst the vessels; so that the bodies of decrescent arteriosclerotics often survive to great and even to extreme old age. In these cases treatment has to concern itself with nutrition and economy rather than with depletion. By adapting the diet to a diminishing output of work, by providing for the best assimilation of the simpler foods, by the addition from time to time of ferruginous and other tonics, by gentle exercises in fresh air, by mild winter climate, and by some relaxation of work and care, but by encouragement of a fair activity of both mind and body and dissuasion from elderly indolence, the tendency to cardiac degeneration or strain is to be counteracted; on the whole by a restorative strategy as patient as the morbid proclivities themselves. Fibrolysins have been suggested, but, in so far as they act as such, they might act injuriously in dissolving the remnant of the framework of the vessels.

Of *toxic arteriosclerosis* the treatment is that of the particular infection or poison concerned, be it in some one or other of the specific fevers, as in syphilis or typhoid; or again in diabetes or plumbism. In the large majority of these cases the blood pressure is not raised, depressor remedies are not required. The most general rule is to make for elimination by rest, with massage baths and diuretics. In early stages vaccines may be required. Free urinary and hepatic action must be encouraged. Every organ must be vigilantly examined, especially the states of the heart and its valves. Except in syphilis, mercury, so valuable in hyperpiesia, is to be eschewed. Even in lead poisoning, often accompanied as it is by high arterial pressures, this drug, save in occasional doses for contingencies, is inappropriate. In cases where a specified vaccine or serum or other antidote is obtainable, as in diphtheria or syphilis, this means will enter into the therapeutical method. But the myocardium is often

affected as much as or more than the arteries. The long survival of microbes, such as the *S. pallida*, in many of these distempers, even after the carrier is wholly or partially immunised, is only too well known. The urine should always be examined bacteriologically. We have, as yet, little knowledge of the ultimate effects of a toxic arteriosclerosis; life may not be so much abbreviated by it as impaired in its energies. We have seen that these influences may tend rather to connective fibrosis than to atherosclerosis. Above all things we shall be warned that in such maladies lie dangers to the heart and great vessels, and thus, foreseeing and vigilant, we shall try, during the acuter stages, to prevent the fixation of these poisons on the tissues. "These foxes must be taken while they are little."

To sum up: involutionary arteriosclerosis is intractable. The toxic forms of arteriosclerosis seem to undergo some mitigation in time; but, syphilis apart, we have few specific means of cure. The use of vaccines may come hereafter; meanwhile we must prevent the engendering of toxins, and promote their excretion. As life goes on, these cases may merge into the involutionary mode. It is in the hyperpietic mode that treatment is most efficacious. If this malady be discovered before it has impressed itself indelibly on the vessels, we may wholly drive it away, or by no very irksome watchfulness dispel it again and again: even if we do not detect it till a later stage, we may still control it, and postpone, if we cannot avert, its extremer perils.

PART II

AORTITIS

ANGINA PECTORIS

SECTION I

AORTITIS

The preceding pages are occupied chiefly by a consideration of arteriosclerosis as a whole, and in respect of the body as a whole. But disease, when seated in the vital thoracic aorta, and especially in the ascending arch, produces some peculiar effects; and if inflammatory, or acutely disintegrating, the issues may be correspondingly grave. Disease thus seated requires therefore some more intimate and special consideration. It was with this object in view that I chose this section of arterial disease for the Cavendish Lecture to the West London Medical Society in 1903.[1] This address, with some additions derived from later experience, and without some parts already included in previous or subsequent pages of this work, I venture to reproduce, as it bears closely upon the succeeding subject of Angina Pectoris (p. 211).

In a little tract of the brain, one so small that an infant might grasp it, lie the ministers of all that makes life worth living; and but a few inches below it, where brain and spine unite, is a still smaller tract, where lies the knot of life itself. The upper tract has its times of energy and of repose, by sleep its ravelled sleave is knitted up; but the knot of life itself knows no pause, no quiescence; let its vigilance be at fault for a few seconds, and the busy frame it governs will drop into silence. Save as a lesson in physiology, these parts are strange to us; yet with it is united another unsleeping minister of life, our familiar, of

[1] The Cavendish Lecture, delivered June 26, 1903. Reported *West London Medical Journal*, July 1903.

whose pulses Harvey was rapt into saying, after Aristotle, that they "are of the spirit of the blood acting superiorly to the powers of the elements . . . and that the soul in this spirit and blood is identical with the essence of the stars." When the pulses of this instrument beat in harmony we feel within us that all is well; when they are jangled and out of tune we are dismayed. Often when in the still night I hear, as I lie, the calm and continual rhythm of my familiar spirit, ever winnowing boon from bane, I am lost in wonder at the long procession of these notes of human time, at this perpetual beat of the manifold tides of life. As I listen to its notes they seem to fall into the burden:

> So schaff' ich am sausenden Webstuhl der Zeit,
> Und wirke der Gottheit lebendiges Kleid.

Sixty times a minute, at least; 3600 times an hour, 86,400 times per day, for us heedful and heedless, does this shuttle of life flit to and fro; for us in tireless periods this pendulum of man's gravitation tells the seconds which will never return.

Tireless as it seems, tireless as sin, yet it will cease; nay, when youth is gone, in every beat there may be an irreparable wound. Hour after hour the blood leaping against the vault of its conduit strains the bonds of it, searching its strength, testing its elasticity. Although, from Professor MacWilliam's researches, it would seem that Roy's estimation of failing aortic elasticity in elderly persons was defective by some ignorance of *post-mortem* and other variables in arterial tissues, yet it is clear enough that some such loss of elasticity is inherent in all men; and in the large majority of aged bodies is in its effects visible to the naked eye. Nevertheless, the endurance of the arch of the aorta under the incessant lashing of the blood for three-quarters of a century is marvellous; in some old persons in whom a constant immunity from toxic influences has coincided with an inherited tenacity of structure, astonishing. I have spoken already of the necropsy of a patient who had died of a gall-stone at the age of eighty-three, how we admired the soundness of the heart, the translucency of its valves, and the uniformity and smoothness of the aorta and its lining. But for the most part it is otherwise, often far otherwise; we expect to find, and usually we do find, in the aorta of persons after the age of fifty no inconspicuous

signs of these years of stress. And, again as we should expect, it is in the arch of the aorta more than in any other arterial tract that these signs of injury prevail, and prevail densely and widely in persons whose lives had been spent in strenuous muscular labour. All this we have considered in the chapters on Atherosclerosis. But, as regards aortitis, with none of its kinds have abnormally high blood pressures any essential concern; if now and then in aortitis they be present it is by accident.

Such being the conditions under which the work of heart and aorta is performed, the stress upon the first or curved portion of the great vessel being thus heavy and perpetual, while we admire its tenacity we shall assume that, to whatever other injurious influences it may be obnoxious, stress must enter for more or for less into the sum of the lesions, and into the election of their seat or seats of greatest severity. Let me suggest a common case; the aorta of a man of some fifty-five years of age, who has taken freely of drink and suffered from syphilis, lies before us. The vessel is heavy, baggy, and hard; its walls are beset with knotty masses. On laying it open we find the stretching had told chiefly upon the ascending arch and vault, and that, besides the old syphilitic pouches and scars, and engrafted upon them, atheromatous bosses and plates also, some calcareous, some "ulcerated," now occupy its inner face. The aortic cusps are thickened and deformed; the sinuses of Valsalva are contracted, and the orifice of the heart is altered. The orifices of the coronary arteries also are invaded by atheroma; the orifice of the right coronary occluded perhaps, that of the left barely admitting a bristle. The cusps, altered as they are, may be competent against regurgitation; the heart in substance looks healthy, and to the naked eye and the microscope, in spite of the interference with the mouths of the coronary arteries, presents in its muscular tissue no worse sign of degradation than a moderate fibrosis. The disease was not cardiac, it was arterial. Here then we find the combined effects of mechanical stress telling upon a vessel formerly attacked by syphilis and further reduced by age and alcohol; to this infection and stress we shall attribute the pouchings of the arch, specific and atheromatous, and so much of the consequences of the mechanical and toxic

lesions as may be due to the incessant agitation of an afflicted organ which, to heal its wounds, needed rest, but to which rest was denied. For this reason disease of the thoracic aorta has always an additional and very grave element of peril. Under the beating of the pulses no lesion, however slight, can be wholly repaired. In favourable cases, by fibroid growth and condensation, a patch of disease may become a patch of repair; for an indefinite time the damaged wall may resist destruction: but *restitutio ad integrum*, or, if the lesion be extensive, even practical efficiency on the computation of an uninjured life, is contrary to experience.

In this case I have spoken of the luetic poison, already fully considered: this however is but one poison out of many by which the integrity of the arch of the aorta may be imperilled. We have seen that the aorta enjoys no immunity from the attacks of the ubiquitous bacterium: although of the kinds and degrees of its liability we are still in some ignorance, yet such lesions may end in atheroma, or directly produce it; but in their acuter stages, the syphilitic, and many other infectious lesions of the aorta, are not arteriosclerosis but inflammatory—are, strictly speaking, aortitis. In syphilis at any rate it ends in scar. Systematic examination of the aorta in respect of infectious lesions, save in syphilis, is much neglected; in infectious diseases, or indeed under any circumstances, careful sections of the ascending arch, not to mention the abdominal limb of the vessel,[1] are very rarely made, or even thought of. Not infrequently I propose the subject to candidates for our higher degrees, but little work of the kind is sent in.

We may anticipate that the self-purifying virtues of the blood and the scour of the aortic stream may give the aorta some advantages against microbic invasion from within; and that, when infection of it occurs, it occurs under conditions of high blood impurity and high microbic or toxic virulence. The microbe may be chased along swifter currents to settle in regions of more sluggish irrigation. We are familiar with it in many cases of malignant endocarditis. Nevertheless, microbic attacks upon the aorta are much more frequent than is generally suspected,

[1] Morgagni always opened the aorta in its full length, and advised that this precaution should not be omitted.

and we have surmised already that passing poisons or toxins, apt to enter into chemical relations with the wall of the vessel, may be the causes of some of that atheromatous decay which we call senile. But, speaking generally, as we have seen, infections attack the vessel by way of its vasa vasorum.

Disease of the aorta has not only a certain independence of disease of the heart, but also of the aortic valve itself. When we speak of diseases of the "aortic area," we mean, no doubt, something more than the arch of the aorta; we include the mouths of the coronary arteries, the valve, the base of the heart; even the large anterior limb of the mitral valve, the smaller being related rather to the auricular side of the heart. We observe accordingly that the larger mitral limb is often associated in disease with the aortic valve, whereby, independently of dilatations, it comes about that a systolic mitral murmur may follow aortic disease; in atheroma it commonly does. Notwithstanding, when we consider the part of the aorta itself, we shall see that disease of the base of the vessel may not invade even its valve; or may involve it gradually, secondarily, and from above. Clinically also, as I shall presently set forth more at length, disease of the arch of the aorta forms a chapter apart from that of the heart.

Aortitis may broadly be divided into Acute and Chronic, a division corresponding with some difference of symptoms, at any rate in respect of intensity, from a course wholly latent, to a more or less transitory stenocardia, to a fuller angina, or to the racking agony of the status anginosus.

More than once I have recalled a case of acute aortitis in which I was concerned when clerk to Dr. Bence Jones at St. George's Hospital, about the year 1860. The symptoms and their origin made an ineffaceable impression on my memory, and from that time forward aortic disease engaged my interest. And as, a few years later, in the diagnosis of disease of this great vessel I became much indebted to the teaching of Professor Potain, I have made from all available sources of observation and reading no inconsiderable a collection of facts. Acute aortitis is not always the perilous disease I once supposed it to be, and it is part of my present purposes to insist upon careful heed to the state of the vessel in diseases wherein aortitis may

enter and depart, often unawares. That acute aortitis is apt to arise in the course of infectious diseases, even in the course of the milder exanthems, rests upon a collection of facts now large enough to carry conviction; though perhaps in the majority of cases it is betrayed by no telling symptom. Incidents of this kind are now no curiosities, but integral, if not common, terms of various series; terms which range in significance from transitory and superficial affections to terrible and even mortal calamities. Acute aortitis, as well as atheroma, was not unknown to the immortal Morgagni; and near our own time, Hodgson, in his notable Jacksonian Essay of 1814 to which I have often referred, carefully distinguished the signs of inflammation from those of imbibition; and, besides his leading description of the aortitis we now know to be syphilitic, described a remarkable case in which a fibrinous deposit lay upon the inner face of the great vessel, and was continued as a thrombus into one of the carotid arteries: a case presumably of bacterial infection.

Causes.—In the infections due to acute aortitis the occurrence of microbes, known or assumed, is beyond cavil. It is far more frequent in the exanthems than we have been wont to suppose. Often mild in degree, transient in time, secret in its approaches, the affection may be revealed, if at all, only to him who is on the watch for it (p. 260). Physicians in charge of **smallpox**, if they will use the means of diagnosis to be discussed hereafter, may detect the lesion not infrequently, or so it would appear from the reports of Therèse, who records six cases, and of other physicians. Brouardel[1] also gives the sanction of his authority to variolous aortitis, and tells us that it arises during the suppurative period, and disappears during convalescence. In some cases it seems to have determined a fatal issue, but often, as it usually appears in cases of gravity, the mortal effects of the aortitis itself cannot be precisely decided. Portal[2] recorded a case of a young man, who died of smallpox "with violent suffocations and palpitations," the wall of whose thoracic aorta was swollen and soft, especially the

[1] Brouardel, *Arch. gén. de méd.*, 1874.

[2] Portal, *Anat. Med.* vol. iii. p. 127; quoted Corrigan, *Dub. Med. Journ.* No. 35.

internal membrane. Happily our experience of smallpox is now very small. In ordinary cases of variolous aortitis the lesion seems to disappear in a few weeks.

It is remarkable that in an infection so trivial as **measles**, if we may judge by the testimony of physicians so well known and careful as Sansom and Dr. Samuel West, the intercurrence of acute aortitis of a mild and transitory kind is not very rare. Of **scarlatinal aortitis** I know nothing, of scarlatinal myocarditis only too much; but we have the authority of Landouzy and Siredey, and of other authors, to testify to its occurrence; and this the more commonly perhaps for the well-known cooperation with scarlet fever of a process very like rheumatic fever, like it not only in its arthritic but also in its endocardial manifestations. Simnitzky of Prague,[1] who worked with Chiari, reckons that aortitis occurs in half the mortal cases of scarlet fever! Both intima and media are attacked. Wiesel[2] includes scarlet fever, with diphtheria, measles, pneumonia, and influenza, among the infections causing aortitis. He warns the pathologist that *in these diseases microscopic evidence of aortitis is often found in cases where to the naked eye the vessel seems normal.* A remarkable case has been published by Minet[3]:—soon after defervescence a fresh rise of temperature occurred with pain in the epigastric portion of the aorta, which dilated and throbbed. There was no other affection of heart or aorta. I shall discuss these cases again later (p. 304). One or two other instances of abdominal aortitis after this infection are on record. The following deserves notice:

Mr. Z., æt. 26, sent to me, April 30, 1908, by Dr. Glasier of Mildenhall. Scarlet fever three years ago; remembers that he had some rheumatism with it, especially in the legs. On rising from bed was seized with severe pain, at first in the subepigastric region, but far more intensely, and afterwards wholly, in the chest. This pain was paroxysmal and intolerable, and for some weeks never went entirely away. It "screwed him through from the breast

[1] Simnitzky, *Zeitschr. f. Heilkunde*, April 1903, and *Bull. de la Soc. de Pédiatrie de Paris*, Nov. 1902.

[2] Wiesel, "Erkrank. art. Gefässe in akut. Infect.," *Zeitschr. f. Heilkunde*, 1906.

[3] Minet, J., "Aortite abdominale, angine post-scarlatineuse. Guérison," *Soc. méd. des hôp.*, juillet 12, 1912.

bone (midsternum) to the back, and went across the left upper chest and down one or both arms (not quite sure about the right)." Does not smoke ; syphilis absolutely denied. The paroxysms were only to be calmed by subcutaneous morphia. To his doctor's surprise after some weeks' illness, and a month's vacation, he recovered completely. Last summer the pains returned, but only for a few days. Last Christmas they again appeared, and he is still subject to them. He is obviously no complainer and presents no sign of " neurosis " ; although no leading questions were asked, he did not readily speak of these facts. His description now is that of classical angina. He grips his waistcoat at midsternum. The only point of peculiarity is that below the elbow the track is that of the musculospiral rather than of the ulnar nerve. Heart enlarged considerably to left, and heaving, but the radial artery, although little thickened, was either so constricted or of such low pressure that in determining the systolic pressure (which was about 110 mm.) only with the greatest pains could I keep in touch with it. (Atheroma of subclavian orifice ?) The aorta was not palpable in the notch, but there was a very strong basal thrill and murmur, rasping and in systolic time. No first sound audible. At the apex a brief and distant first sound was heard with a systolic murmur. No murmur of regurgitation. No Flint sound. No dyspnea, but in attacks sits up with a sense of oppression. As he did not volunteer an apprehension of death I forbore to ask the question.

The poison of **rheumatic fever** prevails of course rather in the mitral area ; but of its power to attack the aortic area, and the aorta itself, we are becoming better aware. In its position in the ascending limb, and in its tendency to aneurysm, it comes nearest to the syphilitic kind. The syphilitic kind grows continually worse, but happily the rheumatic tends to recovery. If in rheumatic fever aortitis may be an extension from the heart, it is not always so. Klotz [1] gives a plate showing a strip of healthy aorta lying, as in syphilis, between a rheumatic valvulitis and an aortitis. Rénon, Klotz, Barié and others testify to the independent position. In 1901 [2] Barié described such a case of aortitis in a child. I have seen not a few cases in which during life it seemed to have an independent seat in the aorta ; and others in which it seemed to be an extension of pericarditis to periaortitis (p. 454). In such extensions

[1] Klotz, O., *Journ. of Path. and Bact.*, Nov. 1913.
[2] Barié, *Soc. méd. des hôp.*, 1901.

of rheumatic inflammation to the aorta, and in rheumatic pericarditis about the base of the heart involving the aorta, as Pawinski [1] and many others have demonstrated, new and strange symptoms may awake in fierce activity.

In my clinical lectures I have been used to make the contrast, to which I have alluded, of rheumatic valvulitis with atheroma in respect of the directions of progress: usually in rheumatism the mitral valve is attacked first, and the disease advances to the aortic valve from the anterior mitral cusp to that aortic cusp with which it is practically continuous; thence it creeps upon the other cusps of that valve, and often extends a little upon the aorta: in atheroma the disease usually extends in the contrary wise, creeping from the aortic valve to the anterior mitral cusp. In the few cases of death during the acute stage of double rheumatic valvulitis, I have seen at the necropsy not only the path of this upward propagation of the inflammation from valve to valve, but also an extension upon the suprasigmoid portion of the aorta; now when this happens we have, in my opinion, the pathological substrate of angina pectoris. Of two cases to be mentioned presently, in that of Dr. Simpson the angina was only too convincing; in the Royston case the process was more stealthily at work; the pain did not transcend the degree of angina minor. After a while these attacks also died out, and this patient likewise, so far as the primary illness was concerned, recovered.

That *in rheumatic fever aortitis is no very rare event* yet nevertheless often an event of no little gravity, is a conclusion which I found, on the delivery of my Cavendish Lecture, was scarcely realised by English physicians. For this reason I renewed the subject before the Association of Physicians, at the London Meeting in 1907. Concerning the rheumatic we have been even more in the dark than we have been in respect of syphilitic aortitis. It occurs not very rarely, but it is unrecognised. Desportes (*loc. cit.*), so far as my reading goes, was the first to describe a case of rheumatic aortitis, in a man, aet. 25, with anginous symptoms; and it is to French physicians that we are indebted for the best of our recent knowledge of it; but Welch also had clearly pointed it out, on pp. 70-74 of his admirable essay.

[1] Pawinski, *Deutsche Arch. f. klin. Med.* Bd. lxiii., 1897.

In most instances it arouses no subjective symptoms; if signs there be they are unsought: in a few cases subjective symptoms are present (*symptômes atténués*) but ignored; as Barié says, "elle pousse souvent inaperçue . . . elle demande à être cherchée."

Among the first recent cases, fully recognised as aortitis, to receive careful clinical study were two published by Marfan (p. 157),[1] in boys aged respectively 12 and 13; and a third, in a girl aet. 13, was published by Méry and Guillemot;[2] but in 1863 H. Roger had published a case of aortitis followed by aneurysm of the ascending aorta in a child aged 10; and in 1881 Hippolite Martin[3] published cases of aortitis in acute rheumatism (as also in smallpox, diphtheria, and typhoid fever). Sanné,[4] in 1887, made such a collection of cases of aortic dilatation and aneurysm as was then possible. I have in my possession an M.D. thesis, of Kiel (in 1891), by Dr. Walther Richter, a pupil of Heller, on the implication of the aorta in endocarditis ("Die Beteiligung der Aorta an endocarditischen Prozessen"). Richter had before him four specimens, and concluded that endocarditis, of whatsoever kind, might invade the aorta in two ways, usually together, sometimes severally; namely, by contiguity or by continuity. Thus an aortic cusp, or a polypoid appendage of it, may, as it flaps, carry infection to a contiguous area of the aorta, where an ulceration may be established from which an aneurysm may arise.[5] Marfan, in a criticism of the relevant physical and clinical signs, brought out certain diagnostic criterions; some of his cases being "trouvailles d'auscultation." He was then of opinion that angina pectoris (by which he meant a full attack with radiating pain and dread) does not occur in children; yet in both his cases there were peculiar recurrent suffocative oppressions and distresses with "douleurs sternales." He also discussed cases drawn from previous authors in which he believed himself justified in diagnosing, or at least suspecting,

[1] Marfan, *Semaine méd.*, mars 27, 1901.

[2] Méry et Guillemot, "Aortite rhumatismale avec dilatation," *Bull. de la Soc. de Pédiatrie de Paris*, Nov. 1902.

[3] Martin, H., *Rev. de méd.*, 1881, vol. i. p. 369.

[4] Sanné, *Rev. mens. des maladies de l'enfance*, fév. 1887.

[5] Professor Teacher showed four or five striking specimens of this kind at the Brit. Med. Assoc. Meeting, Aberdeen, 1914 (see p. 161).

unrecognised aortitis of rheumatic or other infective origin. The alternative was a precocious atheroma, of which a very small number of examples have been recorded from the time of Andral,[1] who noted calcareous atheroma of the aorta in a girl æt. 8, and of Peter, who described a like condition in a child æt. 2.[2] This subject has been well considered by Simnitzky (*loc. cit.*). (See Vol. I. p. 175.)

In the case of aortitis reported by Méry and Guillemot in a child aet. 12, the dilatation of the aorta was considerable and easily defined. It occurred during an attack of acute rheumatism. The "cimier de casque" extended 3 cm. beyond the right border of the manubrium. In 1901 Barie [3] published his similar case with the same diagnosis, and Queille [4] likewise; but they verified rather than increased our knowledge of the subject. In 1903 Zuber [5] reported that he had re-examined one of Marfan's patients at the age of 16. In the previous year signs of general heart failure had set in, with nocturnal attacks of cardiac dyspnea, " and, moreover, during the last year a new trouble, resembling angina pectoris; namely, violent pain in the chest about the sternum and radiating into the shoulder and arms. The aortic dulness extended 4 cm. to the right."

In the same year de la Rüe[6] published a book on the subject. His first two chapters are given to aortic valvular disease; the third to aortitis, which in children he regards as most frequently rheumatic. Now de la Rüe was, I think, the first to point out the febrile phases which may accompany aortitis, as in my Royston case; in illustration of this process he published extracts from temperature charts. The aortitis itself, he says, is apt to bring on pain, radiating to the left shoulder and arm; or passes more covertly as a retrosternal oppression, weight, or constriction. He says also that in rare cases the aortitis may lead to aneurysm. He had seen two instances, of which however

[1] Andral, *Clin. méd.*, 1834. [2] Peter, *Union méd.*, Paris, 1890.
[3] Barié, *Presse médicale*, 1905, No. 24.
[4] Queille, Thèse de Paris, avril 1906.
[5] Zuber, *Rev. mens. des mal. de l'enfance*, Turin, 1903.
[6] De la Rüe, *Affections acquises de l'aorte* (*orifice et crosse*) *chez l'enfant*, Thèse de Paris, 1903, an able tract of 160 pp. De la Rüe says that the first case of aortitis in a child was published by Moutard-Martin in 1875. But this may have been a case of congenital syphilis; and see pp. 151-2.

I gather that Méry's case (reported above) was one. This case de la Rüe verified by radiography. He adds that these aortitic cases may end in sudden death by angina pectoris, or by laryngeal spasm. The author industriously collected, from English and French sources, a very important series of *cases of aneurysmal dilatation and of saccular aneurysm of the aorta in children*; a substantial proportion of which seem to have taken their rise *in rheumatic aortitis*: in some of them *angina pectoris* is definitely described. In these descriptions the author pays the conventional compliments to the coronary arteries. Gibson [1] exhibited a child with double aortic and pulmonary disease—the result of acute rheumatism; she suffered during the attack with pain "which resembled that of angina pectoris." Again, Josué and Solomon [2] in an autopsy on a case of very extensive and acute rheumatic disease, found "on the aorta at the origin of the coronary arteries some *gelatinous patches.*"

In 1905 M. Triboulet,[3] the cordial friend and host of English physicians, published an article on rheumatic aortitis producing cylindroid dilatation of the ascending arch, or of the whole arch; but he had not seen a case of consequent aneurysm. In respect of this consequence however it is interesting to find that Triboulet also detected changes especially in the media; and he reminds us that in Boinet's [4] well-known statistics of aneurysm 7 cases in 240 were attributed to acute rheumatism.

In 1906 (*Edin. Med. Journ.*, June 1906) Dr. Hugh Stewart, then house physician to Dr. Gibson, published a case of severe rheumatic fever in a boy aged 7. The case presented some curious valvular lesions, mitral and double aortic, but incidentally it is reported that two months after the onset, when hardly convalescent, he was seized repeatedly by angina pectoris of the typical characters, radiating into the left arm. "He was fixed in position not daring to move: pale, anxious and fearful." The attacks would last ten to fifteen minutes, and on some days

[1] Gibson, *Med. Chir. Soc.*, March 1903. Also *Dis. of Heart and Aorta*, 1898.
[2] Josué and Solomon, *Lancet*, Nov. 21, 1903.
[3] Triboulet, *Soc. méd. des hôp.*, mars 10, 1905.
[4] Boinet, E., "Aortites," etc., *Tr. de méd.*, Ed. Brouardel, 1907. Idem, *Arch. de méd. expér.*, Sept. 1897; and "Athérome aortique expér.," *Soc. de Biol.*, juin 1906.

were as many as six. The pains did not wholly subside for some two months.

In a volume of *Conférences pratiques sur les maladies du cœur*, etc., published in 1906, M. Rénon [1] has a chapter on Rheumatic aortitis and aneurysm, in which he reports a case of his own in a boy, æt. 16, in the Pitié. The patient was then suffering from acute rheumatism with aortitis. He was seized at the level of the fourth right rib by a sudden violent pain in the breast with "*étouffement syncopale*," but without change of cardiac signs. These attacks were repeated, and radiated to the right arm and shoulder, then subsided with the development of aneurysm. Briefly, the author had the remarkable experience of seeing an aneurysm form under his own eye. It was revealed by radiography. Professor Vaquez saw the case, and agreed in the diagnosis of rheumatic aortitis with formation of aneurysm. The boy had suffered from previous attacks of rheumatism, and rheumatic aortitis had occurred in him before that attack which culminated in the aneurysm. In the next year the following case was recorded in the *Münchener med. Wochenschrift* (1907), p. 2330 :

Male æt. 46 ; good health till the attack of acute rheumatism, on the eighth day of which he had sudden occlusion of the left brachial artery. "Recovery nevertheless complete." But six months later an aneurysm became manifest, with palsy of the left recurrent nerve. It was seen by radioscopy, and proved mortal by rupture. The diagnosis made was Aortitis rheumatica, with embolism of "brachial" artery from the primary patch, and development of aneurysm later.

Bennert [2] has described two cases of aortic aneurysm following rheumatic fever. Both patients were under 20 years of age. Syphilis was definitely excluded. This author had collected twenty cases, and laid down the rule that aneurysm in children and youths is a result of acute rheumatism. Ruppet [3] again reported, in a patient æt. 10, aortic regurgitation and aneurysm of the ascending aorta, due to an attack of rheumatic fever five years

[1] Rénon, *Mal. du cœur*, etc., Paris, 1906; an interesting book in many ways ; also *Presse méd.* vol. xxxviii., 1912.

[2] Bennert, *Zeitschr. f. klin. Med.* Bd. lxix. Nos. 1 and 2.

[3] Ruppet, *Med. Klinik*, 1910.

before. Feytaux[1] reported 5 cases of rheumatic aneurysm in patients æt. 12-16.

Rare then as it may be, aneurysm of rheumatismal origin is definitely to be accepted. As to physical diagnosis little has been added since Potain's well-known lecture in the *Clinique de la Charité*, 1894.

Some authors think that the aortitis is due to inoculation by an affected semilunar cusp, as in malignant endocarditis ; in a few cases it may be so (p. 160), but in a fair number of these cases no valvular disorder was audible. In aortic valvular disease I have observed small patches of inflammation extending from the cusps to the sinuses, and it may travel farther up the arch. But this rheumatic aortitis is but one of many specific kinds, and in syphilis we know the rule to be suprasigmoid aorta first, valvular involvement in the second place. Rheumatic inflammation reaching the aorta from an endocarditis falls into a secondary category.

How, if unsought, this lesion escapes notice I may illustrate by two instances which I discovered accidentally in patients with cardiac disease, due to recent attacks of rheumatic fever, who came in a batch of cases called into Addenbrooke's Hospital for one of our final M.B. examinations. In both besides the heart disease, to one who looked for it, were definite signs of dilated aorta. I brought them under the notice of my colleague Dr. Rolleston, the only examiner who happened then to be disengaged. In neither case had a note been made of this complication ; it was, as usual, lost in the cardiac malady. On the independent notes of one of these, a girl named Jeeves, æt. 15, passed over as aortic regurgitation (and query mitral stenosis ?), I found she had had three attacks of rheumatic fever ; the second "was far the worst." She was then in bed twenty-three weeks, and "suffered severely from attacks of pain at the breast-bone and down her left arm."[2] The other patient, a man æt. 30, also had had three severe attacks, and spent months in bed. In one of the attacks he suffered from severe mid-sternal pain. In both these cases the aorta was much dilated submanubrially, and to right, and up in the neck : throbbing and thrilling.

[1] Feytaux, Thèse de Paris, 1906.

[2] See Appendix ix., I. p. 210.

Marfan,[1] out of twenty-four cases of aortic disease in children, found three cases of aortitis (æt. 9, 12, 13) attributable to rheumatic fever. Many of the others were hereditary syphilis. A very curious case is reported by Hennig,[2] in which aortitis was found in the body of a child born of a mother who during that pregnancy suffered from severe rheumatic fever. Valvulitis may spread by the anterior mitral curtain to the aortic valve, or perhaps the aortic valve may be attacked independently, but I feel sure that in some instances aortic regurgitation, consequent upon rheumatic fever, is due to the extension of an aortitis.

So much for latent cases: the following typical instance will illustrate how slight symptoms of the kind might be neglected by physicians not awake to the process. Master B., æt. 16, a patient of Dr. Windsor of Royston; seen also by Sir Bertrand Dawson and myself. Dr. Windsor, who watched the boy anxiously, wrote, "he should be through a severe attack of rheumatic fever, with a damaged heart, but does not come quite through." He had had several returns of fever, not high but recrudescent; periods which recurred after as before our meeting. In these recrudescences the temperature rose in a flat curve to about 100·5 or 101, or occasionally near 102; and in a few days slowly fell again. Now during these relapses the boy made an anxious if indefinite complaint. He dreaded them; he felt very ill in them, ill beyond discoverable reason; moreover he complained definitely of an oppression about the sternum, so that he would sit up in bed, looking anxious and disturbed. Signs of dilated aorta, dulness and jugular pulsation, were clear and unmistakable. This case was a good example of Leger's rheumatic aortitis "par poussées."[3] I have chosen a fairly obvious instance, but there are plenty of such aortic intercurrencies in rheumatic fever, if less well marked than this one. In a few cases the symptoms, anginous in kind and degree, are actively, even furiously, present, but too often are attributed to the heart, or to transitory spasms of the nerves. In such cases

[1] Marfan, "Lésions acq. de l'orifice aortique et de l'aorte dans l'enfance," *Sem. méd.*, 1901.

[2] Hennig, "Angebor. Aortitis," *Jahrb. f. Kinderheilk.* Bd. xxx.

[3] Leger, *L'Aortite aiguë*, Thèse de Paris, 1877.

the rheumatic aortitis is violent and agonising; angina pectoris appears in full dress, as in the case under Dr. Christian Simpson to which I shall refer again (p. 274). A like case, with necropsy, Dr. Poynton also described in the *Lancet* of May 20, 1899: a woman, aged 38, attacked by rheumatic fever with not only mitral endocarditis but also aortitis (fuller report, p. 275). After death numerous fresh patches were found upon the aorta.

Now although necropsies in cases of rheumatic aortitis are infrequent, as in its acuter stages rheumatic fever is rarely mortal, yet they are less infrequent and more decisive than we suppose. Dr. Andrewes [1] writes: "Professor Klotz informs me that he commonly finds similar changes in acute rheumatism"—similar, that is, to acute aortitis in typhoid fever, scarlet fever, diphtheria, influenza, etc. To Klotz's paper on rheumatic aortitis [2] I have referred already; he likewise describes in the ascending aorta, more rarely in the descending thoracic and abdominal portions, a "fairly typical lesion, after the manner of syphilitic aortitis" (but not syphilitic). The coronary trunks may or may not be affected in this way, though their finer twigs are "invariably" involved in the myocarditis. Klotz gives three necropsies of *consecutive* cases of rheumatic fever in patients whose ages were 9, 16, and 19. The aorta on closer scrutiny presented in each "a striking picture": *the intima was not altered*, but the media was thickened by vascular increase, œdema, and perivascular infiltration, with abundance of leucocytes and plasma cells. Many elastic fibres were "split and feathered," and much muscle had perished. The adventitia was infiltrated by inflammatory non-suppurative lymphoid cells. There were signs of fibrous repair. These histological features were different from the gummy and granulomatous process of lues,[3] though the seat and mode of access were similar. In the older cases patches of sclerosis are formed, chiefly fibrous, but extending to the deeper layers, and with evidence of fatty and even calcareous decay. In one mortal case of rheumatic aortitis and aneurysm, in a child æt. 6,

[1] Andrewes, *Rept. Med. Off. L.G.B.*, 1913, App. B. No. 1, p. 254.

[2] Klotz, *Trans. Ass. Amer. Phys.* vol. xxvii., 1912. But see also the later essay in *Journ. Path. and Bact.*, loc. cit.

[3] The words are those of Klotz; but, as the description is a little abbreviated, I cannot put these sentences as a quotation.

the author found a pure growth of a streptoccocus in short chains. In his later paper (1913) however, he separates two forms: (1) inflammatory hyperplasia *in* the media and adventitia (as if it approached after the manner and by the ways of syphilis); (2) an endarteritis after the obliterans mode. To the first form he attributes the ectasies and aneurysms. Sir William Osler,[1] who was, I think, somewhat sceptical concerning rheumatic aortitis, now agrees that "in rheumatic fever the resulting endaortitis may be serious." Syphilis then is not the only grave toxic invasion of this vessel, nor the only cause of aneurysm, in which, unless the process or its reactions reach the extremer degrees, the intima is primarily unaffected.

Barié,[2] returning to this subject in a recent paper, discusses it as a whole, including peripheral arteritis. This he says occurs more frequently in the arms; in typhoid in the legs. It may occur in the carotid; with fever, pain on palpation, throbbing, and perhaps thrill, with some dilatation. In smaller vessels it may take the obliterative course. In cases of death after bygone rheumatic fever Klotz has shown old thickening of the adventitia and media. He also has demonstrated rheumatic arteritis occasionally in peripheral vessels, as in the posterior tibial.

After my London paper was read, Dr. Cowan of Glasgow sent me a reprint of one of his own,[3] wherein he stated that in acute rheumatism he had found aortitis, and arteritis of its vasa vasorum. In a covering letter he says "the subject (and in other acute infections also) is of far more importance than has hitherto been supposed. In nearly all cases however post-mortem evidence, if obtained at all, is not obtainable till later life, when the incidence of other diseases has vitiated it." Dr. Carey Coombs [4] of Bristol, to certain autopsies of acute rheumatic cardiac disease, adds the following sentence: "At the root of the aorta, about the coronary openings, little white patches are generally visible; these prove on section to be areas of subendothelial inflammation with small-celled infiltrations, proliferative, and fatty change."

[1] Osler, W., *Brit. Med. Journ.* and *Lancet*, Aug. 1908.
[2] Barié, *Paris méd.* tome xxi., 1913.
[3] From *The Practitioner*, March 1906.
[4] Coombs, C., *Brit. Med. Journ.*, Nov. 23, 1907; and *Q. J. Med.*, Oct. 1908.

Pericarditis as a cause of aortitis, acute or chronic, I shall discuss in the essay on Angina Pectoris (p. 454). It is generally a periaortitis due to extension into, or arising in, the cul de sac of the basal pericardium, contiguous to the aorta. It is said that in rheumatic fever this periaortitis may occur as a local and independent effect (see p. 457).

In my essay on Angina Pectoris I shall explain that the presence or absence of sternal oppressions and of radiating pains probably depends—as it does in atheroma—upon the precise seat of the process; whether or no it engages, or at any rate by swelling distends, the investing coat. Now the demonstrations of Carey Coombs, Poynton and Payne, and others, prove that rheumatic carditis is perhaps never confined to a valve, but enters more or less deeply into surrounding tissues; or "it begins in the deeper parts and spreads towards the surface."

From rheumatic fever we pass easily to the **"malignant" forms of cardiac inflammation,** where again we find the aorta by no means outside the sphere of several kinds of virus. In an aortitis which formed part of such a case Dr. Maguire found streptococci. A remarkable case, published by Pagliano,[1] presented the features of malignant endocarditis, but at the necropsy this kind of mischief was found confined wholly to the aorta. Sir Dyce Duckworth has published a case of acute verrucose aortitis; and Boulay[2] also has described a case, in a man aged 36, in whom from a verrucose aortitis, also confined to the thoracic aorta, embolisms in the mesenteric area proved mortal. In three necropsies—two of malignant endocarditis and one of erysipelas—Boinet and Bomary[3] found bacteria in patches of "endo-aortitis." (See Morison, Petit, etc., p. 276.)

Pechter of Kiel, a pupil of Heller, was early in emphasising the extension of malignant endocarditis to surrounding parts, and to independent foci in the aorta.[4] In one case the posterior aorta was much inflamed, and its sinus full of vegetations;

[1] Pagliano, "Aortite chronique terminée par état infectieux sans lésion d'endocarde," *Marseilles méd.*, 1886; quoted in other journals.

[2] Boulay, *Bull. Soc. Anat.*, 1890.

[3] Boinet et Bomary, "Recherches exp. sur les aortites," *Arch. de méd. expér.*, Sept. 1897.

[4] Pechter, *Beteilungen d. Aorta an endocarditischen Prozessen*, Kiel, 1891.

above it were several foci up to the size of a linseed. In a second case the same disease extended to the aortic wall, and had set up ulceration of it. In a third a coagulated deposit, 1 cm. long, was attached to the wall (see Hodgson's Case, p. 148). In a fourth the aortic intima was thickened by proliferation, and in one place about the size of a linseed a superficial ulcer, with ragged edges, and a loss of substance. In a fifth this process was deeper in the wall of the vessel, both media and intima being attacked, with extrusion of the adventitia (clinical notes of this case would have been of much interest, but the author writes only as a morbid anatomist). In a sixth was a small ulcer upon the wall (with aortic valvulitis). In a seventh an endarteritic process had extended to the aorta. Heller himself published a similar case with extension to the aorta, and to the pulmonary artery also.

The verrucose or ulcerative aortitis of malignant endocarditis does not usually tend to perforation; in some cases perforation occurs (see Teacher's specimens, p. 152 *n.*), but the evidence seems to indicate that perforating aortitis is usually a result of other kinds of microbe. In the well-known case of perforative aortitis of Oliver and Woodhead (*Lancet*, 1891) it was the B. anthracis which was detected in the parts attacked. These perforating cases, in which the disease penetrates rather than diffuses itself, are not very rare; though too often the bacteriological evidence is wanting. I shall refer presently (p. 273) to a case of perforation attributed to influenza. So luetic disease sometimes by concentration sets up saccular aneurysm, sometimes is more diffuse. Perforation may make its way into pericardium, pulmonary artery, or auricle; Dr. Lumsden's case of perforative disease of the aorta just above the valve in a young woman of 23, was fatal by penetration and hæmorrhage into the pericardium; the aorta presented only some spots of disease on its inner face. There was no bacteriological record. Thoracic aneurysm of course may take rise in such a process. One of our graduates—Dr. Jordan—has recorded [1] a fatal aneurysm of the ascending aorta in a boy of 6; the aorta and heart seemed otherwise healthy, but a septic focus was

[1] Jordan, A. C., *Lancet*, Feb. 21, 1903. The specimen is in St. Bart.'s Museum with a somewhat similar specimen from a boy of eleven.

discovered in one ear. He had suffered from an arthritis and, presumably, from a septic ulcer of the aorta, due perhaps to septic embolism of vasa vasorum. Nordmann and Maurin [1] publish two cases (æt. 15-17) of pyæmia upon tuberculosis, in which patches of aortitis were formed in the descending aorta. There was no syphilitic infection.

Suppurative aortitis seems to be rare, or, as it does not break into the channel of the vessel, is apt to be overlooked after death. The pus lies in foci in the adventitia and in the substance of the middle coat; it is a periarterial affair, and usually but a small and secondary item in some much more extensive *pyæmia*; as for example in some recorded cases of puerperal infection. Indeed in pyæmia, as in other septic maladies, such an aortitis is not uncommon, and may be due to septic thrombosis or small emboli. Dr. Charlewood Turner [2] has collected cases of this kind; Brouardel and Bureau have recorded others; and at a still earlier date Rokitanski and Lebert had observed both the event and its associations.

Boinet has recorded an instance of aortitis in **erysipelas**, the nature of which he verified by bacteriological tests. Klotz and Sumikawa report that, in some contrast with other infections, the streptococcal set up proliferation of the intima. Staphylococci attack both coats (Saltikow). Klotz says that this infection also enters by the vasa vasorum and, before the intima, attacks the media where, and in the adventitia, small cell infiltration is to be seen. Koritschoner [3] publishes a remarkable case in a man, æt. 30, who died of hæmorrhage by rupture of the aorta into the pleura just posterior to the left subclavian. The cause of the rupture was a perforating phlegmonous inflammation of the media and adventitia, which had arisen "metastatically" from a phlegmon of the hand.

The occurrence of aortitis in **typhoid fever**, as Louis first pointed out, might be recorded more frequently were it more diligently sought for; but in this malady aortitis presents itself in a superficial and transitory form. In typhoid we look for the specific changes rather in the peripheral arteries (see

[1] Quoted in *Zentralbl. f. Herz- u. Gef.-krankhtn.*, March 1912.
[2] Turner, C., *Trans. Path. Soc.*, Lond., 1886.
[3] Koritschoner (Wien), *Zentralbl. f. pathol. Anat.* Bd. xxiii. No. 3, 1912.

Vol. I. p. 283); still Potain has published two cases of definite typhoid aortitis,[1] and a few more are on record. Gilbert and Lion produced typhoid aortitis experimentally,[2] as after them did Chantemesse likewise. The episode, for it is rarely much more, occurs during the later weeks of the fever, or even during early convalescence, when it is very apt to escape observation. [**Scarlet Fever**, see p. 309.]

The occurrence of aortitis in the course of **influenza**, or during the convalescence, has been observed by Lancereaux,[3] by Hanot,[4] by Pawinski,[5] by von Leyden and others; and in England has been so well described by Sansom[6] that I must not linger on this section. (See also Vol. I. p. 291, and Angina Pectoris, p. 268). Some of Sansom's cases came to autopsy, when grey translucent raised patches of aortitis were found. In some necropsies the aortitis had become ulcerative, and in some a mixed infection was verified. Sir Richard Douglas Powell has fully recognised post-influenzal aortitis, often with symptoms of angina pectoris (p. 272). Three or four definite cases, and others of less certainty, have fallen under my own observation; one patient, whom I visited with Dr. Humphry of Cambridge, being an eminent instance. A man, then about 60 years of age and, before his influenza, of excellent health, while apparently convalescent was walking in the fields near his college, when, as I shall narrate in the essay on Angina Pectoris, he was seized in an instant with a severe attack of this malady. He crawled home with great difficulty, and, so intense were the attacks, that he seemed for some weeks to be in hourly jeopardy of life; but ultimately he made a complete recovery, and to this day—many years later—remains well. Kraus[7] says that "grippal aortitis" is not so well known in Germany as it ought to be. He supports also the important rule of prognosis that in this

[1] Quoted in Bureau's *Études sur les aortites*, 1893.

[2] Gilbert et Lion, "Artérites infect. expér.," *Comptes rendus de la Soc. de Biol.*, oct. 12, 1899.

[3] Lancereaux, *Soc. Anat. de Paris*, 1886. See also *Traité d'anat. path.* tome ii. and atlas, 1871; and *Gaz. des hôp.*, juli 6, 1899.

[4] Hanot, *Arch. de méd.*, 1886.

[5] Pawinski, *Berl. klin. Wochenschr.*, 1891.

[6] Sansom, *Roy. Med. Chir. Soc.*, 1894; and *Lancet*, Oct. 21, 1899. See also Fiessinger, *Gaz. méd. de Paris*, Nov. 1892.

[7] Kraus, *Deutsche med. Wochenschr.*, 19. März 1914, p. 578.

kind of aortitis the symptoms and the dilatation usually recede. Sansom records recovery likewise in most of his cases; though others, in which the pathological changes were verified, were mortal. For the records of von Leyden and other observers of this sequel of influenza I must refer to Sansom's paper. Dr. Mitchell Bruce [1] records a case in a man, æt. 54, in whom after influenza angina pectoris appeared. The aorta was dilated. The disease progressed to a mortal issue, when the aortitis was verified by necropsy. In 1908 Marmorstein [2] recorded two notable cases, both in women, æt. 20 and 24 respectively, in whom syphilis could be certainly excluded. In the second patient the aortitis was betrayed by a disagreeable sensation referred to the upper sternum; it was observed to extend to the aortic valve, first with a systolic, then with a diastolic murmur, which were permanent. The former patient died of basilar thrombosis: within the arch of the aorta were patches of acute inflammation, showing abundant cell proliferation, here chiefly in the intima, but penetrating to the middle and outer coats. The vasa vasis were injected and infiltrated around with small cells. *Influenza bacilli were found definitely here*, but more abundantly in the cerebral tissues. (See also Cooper, p. 273.)

Of aortitis following **diphtheria** many instances are on record.[3] This infection attacks the media distinctively (Klotz), a simple medial necrosis; not with a proliferation of the intima, as in typhoid, streptococcal, etc., aortitis. But we may agree with Marchand that it is not infrequently impossible to discriminate between an acute aortitic atherosclerosis and mixed conditions. Curschmann [4] describes such a case in a boy, æt. 16, who died after severe diphtheria of five weeks' duration. The front wall of the aorta was thin and pouched, and its inner face was beset with a network of whitish streaks of disease. The heart and other vessels were examined, by the naked eye and by the microscope, but no changes were found in them. I will stay to cite only one more case of diphtheritic aortitis, in a horse, examined by Boinet, which had been poisoned by a pro-

[1] Bruce, M., *Lumleian Lect.*, Lect. III.

[2] Marmorstein, *Rev. d. méd.*, 10 mars 1908.

[3] *E.g.* Mollard et Regaud, "Ath. de l'aorte avec diphthérie," *Soc. de Biol.*, juillet 17, 1897; and Klotz later.

[4] Curschmann, *Pathol. d. Kreislaufs. Art. med. klin. zu Leipzig*, 1893.

longed and severe course of injections of diphtheria toxin ; acute aortitis, of which illustrative microscopic sections were published by the author, was found at the necropsy.

That aortitis may arise in **pneumonia,** not as a direct extension of this disease, but as a several function of the pneumococcus, from certain observed cases seems probable. Dr. Poynton[1] narrates a well-marked case, in the main a severe pneumonia ; but after death marks of a separate point of attack were found in the first portion of the aorta. The endothelium was destroyed, and the subintima infiltrated with leucocytes. Luzzatto[2] also narrates a case in a man, æt. 45, ill with tedious chronic pneumonia for twelve months, when suddenly an ulcer of the ascending aorta, 1 cm. wide, perforated. The intima was thickened and split, and the media " sclerosed," besides hyaline foci of softening in the muscularis, and abscesses. The elastic fibres had perished. Pneumococci were abundant in the affected parts. The mischief seemed chronic and of some duration, and as if due to direct attack of the diplococci from the blood stream. Barié (*loc. cit.*) has found the pneumococcus in the wall of the aorta.

Malarious aortitis, which attacks the arch especially, does not rest upon the well-known testimony of Lancereaux alone ; Hervé[3] published three cases, and Sansom, adding the weight of his authority to theirs, recorded another in a lady in whom gout, rheumatism, syphilis, and other known causes could be excluded. Nevertheless, as this form of aortitis is not always benignant or transitory but, as Sansom points out, is apt to linger for years and to leave permanent lesion of vessel or valve behind it, we may be thankful for its apparent infrequency. Dr. Theodore Fisher,[4] late of Bristol, notes however that among men who have frequented tropical lands disease of the aorta is prevalent. He thinks syphilis insufficient to account altogether for this prevalence, and suggests that it may be due in part " to some infection of the blood which is more common in warmer climates than with us." Eight of thirty-seven collected cases were attended with the formation of aneurysm.

[1] Poynton, *Heart Dis.*, p. 273.
[2] Luzzatto, A. R., *Riv. d. sci. med.* vol. lii., 1912.
[3] Hervé, " Symptômes de l'aortite chron.," Thèse de Montpellier, 1885.
[4] Fisher, Theo., *Brit. Med. Journ.*, Feb. 21, 1903.

Lancereaux, as we shall see (p. 403), spoke of angina pectoris as an eminent feature of the disease;[1] and Dumolard[2] and fellow-workers have brought testimony from recent experience in support of his opinion. In malarious districts, in syphilis-free individuals, in both sexes, and often before the age of 30, they found dilated aorta, and even aneurysm. They cited only cases in which the Wa.R. was negative. In diagnosis they relied upon the X-rays; but they had one autopsy, in an Arab, æt 20, who died of malaria. The internal tunic was affected, not the adventitia nor media. But the description of the intimal disease, so far as quoted, was not convincing; it seems no more than Hollis, and many others, have described in young subjects.

Tuberculosis of the aorta has been studied by Flexner,[3] Hanot, Blumer,[4] Boulay (*loc. cit.*), Faber (*loc. cit.*), and others. Tubercle, only too common in small arteries, especially in miliary form, as in those of the brain, lung, or kidney, is rarer in the large; and in the aorta its presence is rather curious than important. Its patches are distinguished from syphilitic patches partly by their greater propensity to caseation; the discrete nodules are most frequent in acute miliary tuberculosis. The epithelium and adventitia long remain intact, the media undergoes necrosis. Woolley[5] collected fourteen cases. Little nodules may be found on the inner surface of the vessel not continuous with tuberculous foci elsewhere; or the aortic disease may form part of a chronic tuberculous mediastinitis. In the discrete foci tubercle bacilli are to be found. Blumer readily demonstrated the specific bacillus in both his cases.

Brodier and Laroche[6] have published two cases in which **gonorrhœal aortitis** was almost surely diagnosed. In both a dilatation of the vessel receded with the subsidence of the acute attack. Probably in many of these infections aortitis (as arteritis elsewhere and phlebitis, with thrombosis) is no

[1] I am indebted to Dr. Francis Hare for the following references on this subject: Broadbent, *Lumleian Lect.*, 1891, rep. *Brit. Med. Journ.*, p. 748 (see also *Heart Dis.*, 1807); and Anstie, *Neuralgia and its Counterfeits*, 1871, p. 74.

[2] Dumolard and others, *Revue méd. d'Algérie*, déc. 2, 1913; quoted in other journals.

[3] Flexner, "Tuberculosis of the Aorta," *Bull. Johns Hopkins Hosp.*, 1891.

[4] Blumer, *Amer. Journ. Med. Sci.*, 1900.

[5] Woolley, *Johns Hopkins Hosp. Bull.*, March 1911.

[6] Brodier et Laroche, *Gaz. des hôp.*, mai 22, 1900.

uncommon occurrence, but is latent, or masked by the general symptoms. On this subject Wiesel (*loc. cit.*) has made some interesting observations.

Syphilitic aortitis I have considered in the essay on Arteriosclerosis, and shall have to deal with it again in the essay on Angina Pectoris; but here it must take that chief place which the gravity of the disease imposes upon us. Syphilitic disease of the aorta is far more common than of the heart. Deneke[1] reckons that in adults syphilis doubles the mortality of diseases of the circulatory system; that the aorta is the most frequent seat of the infection, and that in its tale of deaths it is exceeded only by General Paralysis. The inflammation in syphilis is usually chronic; though some cases of syphilitic aortitis are of rapid course, and may do more grievous, or swifter, injury than the more gradual phases of extensive chronic disease and dilatation. Many years ago Dr. Pye-Smith put such a case on record, in a syphilitic subject, æt. 32, in whom was no history of rheumatism or chorea. At the necropsy a patch of recent aortitis was found; soft, injected, and crescentic at the edges like a cutaneous syphilide; it had advanced rapidly, invading and destroying the valve. Brault, quoted by Letulle,[2] examined a fresh and acute syphilitic patch which will be referred to presently in the pathological paragraphs of this chapter. In the essay on Arteriosclerosis I have described how *syphilitic aortitis passes into or blends with atheroma*, or may even be concealed by it; in persons of and after middle life, as Welch pointed out, we have no clear local demarcation—in many cases at any rate—between the syphilitic and the atheromatous processes. Hodgson, Wilks, Moxon, Davidson, Jaccoud,[3] Potain,[4] and many others taught us much about syphilitic aortitis; but it is to Francis H. Welch, sometime Professor of Pathology at Netley, that we owe our full knowledge of this subject. It cannot be too plainly declared, in these days of "Doehle-Hellersche Aortitis," that Welch, in his comprehensive

[1] Deneke, Th., of Hamburg, "Die syphil. Aortenerkrankung," *Deutsche med. Wochenschr.*, 1913, p. 441. Seaport statistics are over the average.

[2] Letulle, *Anat. path.*, 1897.

[3] Jaccoud, "Aortite et aneurysme de l'aorte d'origine syphilitique," *Sem. méd.*, 1887.

[4] Potain, "De l'aortite," *Union méd.*, Paris, 1889; also Charcot, *Œuvres complètes*, tome x.

and judicious essay, if in places a little old-fashioned in expression, described nevertheless all the essential features of the process, macroscopic and microscopic. In November 1875 [1] he read his paper upon 117 cases of "fibroid aortitis"; in 46 per cent of which was a clear record of syphilis. Again, of a series of 56 cases of fatal syphilis, 34 presented aortic lesions, mostly severe. If extensive, it may produce Hodgson's dilatation; if circumscribed and penetrative, aneurysm. Welch's opinions were strongly contested at the time in the Royal Medical and Chirurgical Society, and later, through his pupil Orth, by von Leyden, and even by Virchow himself. Herein Orth's pupil Bruhns must have differed from his master, for Deneke says Bruhns was the first to describe a congenital case.

In discussing the syphilitic aorta I shall pass by, for the most part, the large subject of aneurysm; my concern is rather with those parietal changes of which aneurysm, if a very obtrusive, is an incidental offspring. Hodgson, in the admirable description, in his Jacksonian Essay of 1814, of this præternatural dilatation of the ascending aorta, beginning just above the semilunar valve, seems to me to have suspected syphilis as the cause of it (see pp. 9-10); and he quotes, of those before him, Morgagni, Senac, Lieutaud, Haller, and Scarpa; now Scarpa clearly apprehended the specific cause as Lancisi had done. Scarpa says, "*Syphilitic patients are very liable to steatomatous* [an unlucky adjective] *and ulcerative disease of the coats of the aorta.*" He gives a case, with aneurysm, in a corporal, æt. 22, who had been treated with mercury; though he was disposed to ascribe this effect rather to the mercury than to the syphilis itself. But, as Hodgson says, these authors confused this chronic aortitis with aneurysm. In the original and still unforgotten sense of the word aneurysm they had their excuse; Welch in his classical paper compares fusiform and saccular aneurysm. Hodgson did good service moreover in emphasising clearly that post-mortem imbibition was not disease. Other authors contended, not that syphilis set up its own specific process, but that it gave a bent to ordinary atheroma. Now we know that of mere atheroma aneurysm is a very rare consequence; indeed the great Neapolitan surgeon associated what he definitely named as the "Aneurysma Gallicum"

[1] Welch, F. H., *Med.-Chir. Trans.* 2nd ser. vol. xli., 1876.

with "impure coitus" and signs of syphilis elsewhere in the body;[1] and attributed the aortic lesion to the "principia ichorosa" and "humores erodientes" of it in the blood; though he is puzzled to know how, while in the current, they can fasten upon the wall. Rayer[2] likewise was convinced that syphilis played a part in this aortic lesion; and later Gueneau de Mussy, whom I often find reason to quote, published, in the first volume of his *Clinical Medicine*, an admirable study of the subject, with its pathology well up to that date. Edgren[3] was clear about the lues, but was not clear about the peculiar histology; even my friend von Schrötter never quite saw his way to accept this distinction. Sir William Osler[4] says that Helmstädler[5] drew attention to the rupture and necrosis of the elastic fibres, causing grooves, which he pictured; but this author again did not realise the specific nature of the lesion he had under his eye. For I may note that, thus far of course, this process is not necessarily specific. Osler adds that Köster, in 1875, stated that "mesarteritis" is a mark of syphilis—which is true, if not critical, for the same is true of rheumatic and other kinds of infectious aortitis originating by way of the vasa vasorum—and that in 1877 "gummous" deposits were recognised in the media. We have seen that it is difficult, save in a few cases, to assure oneself by the eye of the "gummous" nature of these foci (Vol. I. p. 300 and II. 184). Eppinger's work on aneurysm (in 1887) Osler esteems as "one of the most exhaustive ever written." In the same year Jaccoud[6] described a case of syphilitic aortitis; and I well remember that about these years, or sooner, my old comrade Dr. W. H. Dickinson was demonstrating to us "syphilitic atheroma." Great progress then was made; albeit the syphilitic was not yet clearly distinguished from the atheromatous process, which, as I have said, practically never leads to aneurysm.

We shall not forget what is due to Heller (1885), to Döhle (Inaug. Diss., Kiel, 1885), or to Saathoff; nor our earlier debt to

[1] Lancisi, *De novissime observatis abscessibus*, c. xviii., ed. 1724.
[2] Rayer, *Arch. gén. de méd.*, 1823.
[3] Edgren, *Arteriosklerose*, Leipzig, 1898.
[4] Osler, Sir W., Schorstein Lecture, *Brit. Med. Journ.*, No. 27, 1909.
[5] Helmstädler, *Formation des anévrysmes spontanées*, Strasburg, 1873.
[6] Jaccoud, "Aortite et aneurysme syph.," *Sem. méd.*, Paris, 1887. See also Faure, *Arch. gén. de méd.*, 1874; Kundrat, "Aortitis acuta," *Allg. Wien. med. Zeitung*, 1885; and Broadbent, *Heart Diseases*, 1897.

the Scandinavian physicians—to Heiberg (1876) and to Malmsten; to Benda again about 1888, and later to Chiari.[1] It is not surprising that our more intimate knowledge of syphilitic aortitis has come from great seaports, such as Kiel, Hamburg, and Christiania. Reuter of Hamburg (in 1906) was perhaps the first to find the spirochæta in these cases. It is however to English medicine, to Hodgson and especially to Welch, that we owe our knowledge of this disease, debts too much ignored by German writers who, too exclusively, claim the honour of its description for the Kiel School; and our debt herein is not much less on the whole to Wilks, and his pupil Moxon.

Professor O'Sullivan [2] has graphically described, in two cases, the features of this characteristic form of aortitis. Some years ago he had received the heart of a young soldier who had dropped dead on the march. He was apparently perfectly healthy. The appearance of the aorta struck him as abnormal, for he observed that a little above the origin of the aorta, parallel with the valves, there was an abrupt rise in the surface level which continued in breadth for about an inch upwards into the vessel and then as abruptly descended again, forming an elevated partial ring, a quarter of an inch or more higher than the rest of the surface. The left coronary artery was obstructed by the excrescence of the surface, which obstruction he took to be the cause of death. That specimen had gone astray, but he exhibited a similar specimen from a young man who a few days before had been found dead in a lavatory. The condition of the part in the two cases was almost identical. There was very little degeneration, as compared with an ordinary case of atheroma; and the amount of overgrowth of the inner coat of the vessel, as compared with the amount of degeneration, was enormous. The orifice of the right coronary artery was practically closed; the opening of the left was likewise reduced to small dimensions. A small depressed area, about an inch from the sinuses, was stained red and exhibited a lesion different from atheroma. The tumidity of the inner coat, as seen under the microscope, was composed of a reduplication of the inner

[1] Chiari, many papers: the earlier in discussions of Deutsche path. Ges. in 1899 and 1903-4.

[2] O'Sullivan, Session Roy. Acad. Med. Ireland. Rep. *Lancet*, April 11, 1908.

tissue; in the middle coat the elastic tissue was cut, torn, and split by masses of cell infiltration which ran along the course of the vasa vasorum. The natural conclusion was that, although there was no direct evidence of it, both cases were syphilitic.

But the weight of the incidence of syphilis upon the aorta and its valve is even yet not fully recognised. Oberndorfer,[1] in a recent discussion, reported that of 1436 autopsies on adult bodies during the previous two and a half years, "Aortenlues" was found in 99—nearly 7 per cent—and that it stood next in frequency to diseases of the heart. In 40 of these cases the aortic disease was discovered at the necropsy. He urged the importance of suspecting syphilis in all slow and obscure cases of disease of the heart or aorta. He emphasised *the larger pulse amplitude* (dilated aorta), the (occasional) angina, and the usually normal systolic pressure, unless of course the valve be injured and the left ventricle hypertrophied. Gruber of Strassburg,[2] who followed this speaker, stated that he had searched the records of 6000 cases, and found luetic aortitis in 4 per cent; or, taking cases since the vogue of the Wa.R., 4·5 per cent. I may say here that with expertness in the Wa.R. method, the proportion of positive results is increasing. Of 256 autopsies on bodies with acquired syphilis in Leipzig, between 1906 and 1911, Marchand reported aortic syphilis in 211 (about 82 per cent); and in 67·4 per cent of autopsies on the congenital disease; in but few of these was the pulmonary artery the vessel attacked. So large is the post-mortem incidence that Marchand suspects that in the slighter invasions the local disease peters out more or less harmlessly. Citron[3] used the Wa. test in all chronic cases of aortic regurgitation, and found it positive in over 60 per cent, a much larger percentage than that of a history of infection; and recently even this estimate has been exceeded. Deneke, in cases

[1] Oberndorfer, E., *Münchener ärzt. Verein*, Nov. 20, 1912, reported in *Münch. med. Wochenschr.* Bd. x. S. 506, 1913; report contains precise records of morbid histology. See also idem, *Berl. klin. Wochenschr.* Bd. xlix., 1912.

[2] Gruber has since published a monograph on "Döhle-Hellersche" Aortitis (1914), too late for this book. It is rich in pathological material, and contains some important statistics. It is well illustrated. But in the results there is nothing new.

[3] Citron, *Berl. klin. Wochenschr.*, 1908.

apparently specific, is still improving upon his positive percentage of 88·6. Out of 71 pronounced (*ausgesprochenen*) cases of aortic syphilis Oberndorfer (*loc. cit.*) reported a positive Wa.R. in 67. Collins and Sachs [1] tested 13 cases of aortic valvular disease sole: in 10 the reaction was pronounced, and in one more it was apparently positive; these cases they contrasted with an equal number of mitral disease only, in which the reaction was positive but twice. Allen [2] however obtained a positive Wa.R. in some cases of mitral stenosis, in which the fibrous circumference of the orifice proved to be thickened by syphilis, so that we cannot confine our suspicions to aortic disease; in a few cases of mitral stenosis of obscure origin the same infection may be at work. Indeed we should bear in mind the possibility of an element of syphilis in cases of heart disease apparently of other causation, for by a positive Wa.R. we may be directed occasionally to find no little aid from the addition of specific to other remedies.

Oberndorfer (*loc. cit.*) has suggested that a syphilitic spot might give a starting-point to a verrucose or other septic cardio-aortitis.

As regards *sex*, aortic syphilis occurs, according to the records, more frequently in men than in women; but this proportion, being about three to one, probably depends on more than one factor other than the intrinsically sexual. Allen (*loc. cit.*), while reporting the heavy prevalence of syphilis in Victoria, found there the proportion of this aortic lesion in women relatively large. In 1906 he had 31 deaths from aneurysm in women, as against 57 in men; and he found a similar unusual relation of aortic insufficiency in the two sexes. Of 173 cases in Deneke's report (*loc. cit.*) of his previous four years, 148 were men, and only 24 women (the remaining one was a congenital case). In one-half of Benary's [3] cases the aortic disease was the only manifestation of syphilis; concomitant symptoms were most frequently the "parasyphilitic." In one-third of them some tabi-paralytic symptom was detected. There was valvular (aortic ?) lesion in one-half

[1] Collins and Sachs, *Amer. Journ. Med. Sci.*, Sept. 1909.
[2] Allen, *Intercol. Med. Journ. of Australia*, March 1909.
[3] Benary, a Kiel student, Diss. Kiel, 1912.

of them. *A frequent symptom was angina pectoris.* Stadler reports a case of aortic syphilis in a widow whose husband was syphilitic, and likewise had this aortic disease. When I read this story it flashed upon my mind that I had seen the same consequence. The husband was syphilitic, and both he and I feared his wife had become infected; she underwent a course of specific treatment, and we hoped the matter, equivocal at worst, was at an end. After her husband's death, when she was under 60, she came to me for angina minor with aortic dilatation; the angina slowly increased, and caused her death.

In respect of *age* the average falls between 35 and 50; much earlier than atheroma. Like tuberculosis, the disease mars and kills its victims in the prime of life. Benary, of 54 cases of syphilitic aortitis, found the mean age to be 48·6; the mean period after infection 22·9 years. The majority of these patients die between the ages of 40 and 50 (Lippmann, Deneke, Stadler, etc.); and no small number present symptoms between the years of 30 and 40. Sir W. Osler, in his Schorstein Lecture, reports two cases of patients aged about 21; and Lippmann (p. 181, note 5) a case of a young man, æt. 20, who had been infected very soon after (possibly before) birth. His father was syphilitic. Benda [1] has described a case in a woman, æt. 22, who died on the third day of acute anginous symptoms: P.M. moderate aortic insufficiency, and starting from the first portion of the aorta a severe scarring (*schwielenbildend*) inflammatory process, with dense thickening about the root of the aorta, suggestive of gumma; and of this nature under the microscope it proved to be. Benda showed the sections with the lantern. (See also Case II. p. 375.)

Congenital cases of this disease, noted at such ages as 15 or 20, are not infrequent. Rebaud [2] reported an examination of the aorta in 17 syphilitic fœtuses and infants. In 13 he found specific aortic disease, and in 5 of them the spirochæta. He found most in one case, a severe one, in which the mischief had extended to the intima. Marchand has reported a case in a virgin, a subject of inherited syphilis, in whom aortic lues did

[1] Benda, at a meeting of the Berlin Medical Society on Jan. 19, 1910: protocol in German journals about date; also in Aschoff's *Lehrbuch*, Bd. ii.

[2] Rebaud, *Münch. Monatsschr. f. Geburtsh. u. Gyn.*, July 1912; and *Berl. klin. Wochenschr.*, Aug. 5, 1912.

not reveal itself until the age of 27; and Ziegler in another case recorded a like protraction. In many cases of young children dying of congenital syphilis some measure of this peculiar affection may be found on scrutiny of the aorta (Heller, Chiari). Klotz (*loc. cit.*) and others have dwelt fully on this part of the subject (see also Vol. I. pp. 174 and 293).

As this aortic disease, before it descends upon the valve, is often latent, we are deprived of many data and of many opportunities: for example, we cannot say *how long an interval elapses, or may elapse, between the primary infection and the aortic lesion.* Probably the interval is very variable, and the extremes wide apart. Aortic disease has been recorded so early as eighteen months after the primary infection, and so late as forty years after it; though in these belated examples the aortic mischief had probably lain latent for years. A. Fränkel [1] quotes a case of "cardiovascular syphilis" two years after infection. To illustrate this latency I might refer to many a necropsy on cases of tabes and of general paralysis of the insane; for in such cases Hodgson's aorta, in its various degrees, is no uncommon *trouvaille d'autopsie.*[2] It is, as I have said, because of this latency of the early stages that the period between primary infection and aortic lesion is so difficult to calculate. Estimates vary widely because the intervals vary widely. In two cases of Stadler's,[3] only five years elapsed between infection and the first symptoms of aortitis. Brooks [4] relates two cases in which aortitis appeared with early secondary symptoms (roseola, etc.). The speakers at the Inner Medicine Congress at Berlin in 1914 agreed that the intervals might vary from 2 to 5, 13, and 39 years. Fränkel said the aortitis might appear "very early," or be deferred 20-30 years. In a coloured picture of a section in Letulle's *Path. Anat.* (1897), which shows subintimal as well as medial and adventitial lesions, the age of the patient is not given, but the whole process seems contemporaneous. In one of my cases the infection was

[1] Fränkel, A., *Berl. klin. Wochenschr.*, 1913, No. 17. See also *Deutsche med. Wochenschr.*, 1908, No. 12, and *Münch. med. Wochenschr.*, 1908 and 1912.

[2] So far as my notes go, the first pathologist to observe, but not at that date to explain, the concurrence of aortitis with general paralysis was Bordès-Pages, in a Thèse de Paris of 1885, in my collection.

[3] Stadler, *Die Klin. d. syph. Aortenerk.*, Jena, 1912.

[4] Brooks, *Amer. Journ. Med. Sci.*, Oct. 1913.

at the age of 18; the aortic symptoms appeared about the age of 41 or 42 ("æt. 43 to 45," Deneke); death occurred at 51. (See also case, p. 516.) At Milbank, of 44 cases in soldiers 15 were under 30; 36 under 40 (quoted by Osler). It seems that intervals between twenty and thirty years are not infrequent. Lippmann notes intervals of fifteen to twenty years after infection; Deneke a mean interval of twenty years, with extremes of five and forty-four years. Osler quotes a case in a man, æt. 36, in which aneurysm of one coronary artery appeared only eighteen months after the primary infection. Lubarsch, in 1911, reported two cases of specific cerebral symptoms in which this arteritis occurred in seven and fifteen months after infection respectively. Data of this order are sought from the point of view of "tertiary" *versus* "parasyphilitic" phases; and to ascertain whether, if "parasyphilitic," specific treatment be effectual. The frequent association of aortitis with tabes and general paralysis does not by any means necessarily pass on the lesion to a "parasyphilitic" category, if such there be.

Occupation and other circumstantial determinants, if any, are not known. By my own experience I should be led to say with some decision that laborious or athletic pursuits are important determinants. Deneke and others do not regard alcohol as an important determinant, and I agree with them. It so happened that few, or none, of my many cases in private practice were in sots; two or three were in abstainers.

Two cardinal methods of diagnosis have opened out to us almost a new field of diagnosis in aortic disease, *the X-rays and the serum test*; devices by means of which we are extending and confirming our data very largely indeed. As concerns the lesion under discussion, they aid us to determine its seat, its nature, and its extent; great gains, if not all we could desire.

The *seat of the mischief* in the suprasigmoid area, in its first stage a single cushion less than a sixpence, is a matter of the gravest import, because *it threatens* precious limbs of the arterial system—*the aortic valve, and, at their orifices, the coronary arteries.* It is therefore of the utmost urgency to discover, or even to surmise, the initiation of such an evil, which, "working on in its still design," will wreck the frame on which it has made

its settlement. Of late years I have had the satisfaction of believing that by prompt diagnosis, and prompt treatment, I have been able to save from invasion the valve—which however may be distorted by a peri-cicatricial process, the cusps being still of fairly normal quality—and probably, as results have suggested, the coronary orifices also. For we shall see that cases of *syphilitic aortic regurgitation*, perhaps crippled because of this common association of closed coronary orifices and aorta, do on the whole badly, very badly—far worse than rheumatic cases of equal reflux; although, as Grau points out, in rheumatism the vascular signs are usually far more salient.[1] In syphilis the left ventricle is less reinforced, and the enormously dilated aorta fails as a bellows. We know that atheroma rarely engages with the aortic orifice in such a way as to cause regurgitation; Stadler indeed makes the surprising statement that for nine years in his clinic (8000 patients a year) he had met with no case of aortic regurgitation due to (non-syphilitic) atheroma; he infers that aortic regurgitation in the elderly, if not rheumatic, will prove by the Wa.R. to be syphilitic. But to regard every lesion in a Wa.R. positive patient as syphilitic is surely rash assumption. Still it is true that the effect of atheroma is rather towards stenosis; of syphilis to a dilapidation of the passage.[2] Apart from the incident of aneurysm, or of angina, syphilitic aortitis which has advanced to this point, which has broken open the gateway, forced the gates, and blocked the sources of supply, may bring life to an end within two or three years, as in the case of the fine powerful young man under the care of Dr. M'Ilwaine (p. 179); whereas aortic regurgitation due to other causes, such as bygone rheumatism, may be consistent with a long survival—even of twenty years or more. Unhappily in the syphilitic case, so covert in its approaches, only too often we are not consulted until regurgitation is established; too commonly indeed the sounds of regurgitation are among the first signs to be noted; the worst mischief is then virtually

[1] See, for example, an interesting case published by A. Morison, *Lancet*, Feb. 3, 1912; and a paper by Fortescue Fox, *Lancet*, July 8, 1911, p. 73.

[2] In one or two cases of my own after a few (three or four ?) years the regurgitant murmur ceased; the systolic continuing alone. This alteration I guessed might be due to a contraction of peri-aortic scar tissue; but I had no autopsy. The patients certainly seemed to be at some better advantage.

achieved. We cannot be sure whether or no in a particular case the coronary arteries are blocked; but on common experience the forecast is gloomy enough.

If regurgitation do not ensue the prospect is brighter; but, as we have seen, the latency of aortic syphilis deprives us of data for its average duration. The appearance of a regurgitant murmur gives us indeed a starting-point, but for dissolution, not for initiation; aortitis is an advancing lesion, but at what rate of advance, past or future, we cannot compute. Deneke found that of his 178 cases, rheumatism being carefully excluded, in 124 the syphilis invaded the valve; and of these in 46 both aortic regurgitation and aneurysm were present together. Until regurgitation appears however we are not obliged to give, or to apprehend, the gravest prognosis. Saathoff,[1] it is true, found that in six out of seven cases this aortitis had led to regurgitation, but Deneke's and Saathoff's figures, if valid for cases in sailors and dwellers about a seaport, many of them virulent and neglected, are too high for ordinary mixed practice. We know that a considerable proportion of cases of aneurysm do not extend to valvular insufficiency; and, in and out of hospital and my own consulting rooms, I recall case after case of aortic syphilis arrested, by nature or art, before audible impairment of the valve.[2] In one case of Hodgson's disease, long under my own observation, one in which the aortic valve never became incompetent, though for some ten years signs of aortic dilatation, which for all this period were obvious, slowly increased, life was prolonged until the age of fifty years, and for much even of this period in health and activity. Death was by agonising seizures of laryngeal spasm. This patient travelled much about Europe, and in Egypt; and it was curious to find how few of the many eminent medical men whom, here and there, at home and abroad, he consulted, recognised the true nature of the case. Scarcely any of them

[1] Saathoff, *Münch. med. Wochenschr.*, 1906 and 1910.

[2] Gruber found aneurysm in about 20 to 25 per cent of aortic syphilis, and computed that, of the cases of aneurysm of the thoracic aorta, aortic regurgitation appeared sooner or later in about one-third. These proportions for my experience seem both of them high. By the way—to digress to the visceral arteries, such as the splenic or mesenteric—we may find in them every stage of the formation of saccular aneurysm, from incipient slight ulceration to deeper penetration and formation of sac *with clean edges.*

then seemed to be familiar with the Hodgson-Welch aorta, a state which ultimately was fully demonstrated by necropsy. In this case an eminent person, eminent in surgery rather than in medicine, was saved only by chance from operating for innominate aneurysm! So long then as the aortic valve is competent, we are justified in hoping for a conservation of life for some years; and perhaps, by active treatment, for an arrest, or staving off, of the affection. I cannot therefore agree with the reckoning of a recent distinguished author that after aortic syphilis is established, the mean duration of life is only one and three quarter years. Unless the numbers are very large, a few swift cases may bring down the mean to a very low figure. Such means, without statement of extremes, are worse than useless.

When, if at all, the coronary orifices become involved we cannot tell. But I have said that in the cases of syphilitic aortic disease with regurgitation the prognosis is unfavourable. Grau [1] and Stadler [2] also have rightly laid great emphasis upon the default in this lesion of a due and stable hypertrophy of the left ventricle; Grau goes so far as to say boldly that if the compensation be good the case is not lues. This maxim corresponds with my own experience, though in a syphilitic case now under my care a fair compensation has been attained. In such cases the coronary arteries may have escaped; as, on the other hand, they are often blocked without valvular incompetence. In regurgitation from other causes, the heart, as we all familiarly know, grows vigorously up to its work, and, if the reflux be not intolerably copious, will for many a year keep up no very inadequate balance of function; but in syphilitic disease, as a rule, the insufficiency is not met in this definite and energetic way. These far from uncommon cases do badly. Although after death the parts may not indicate a large regurgitation the heart does not rise to the occasion, or does so imperfectly. I recollect one case in which after death the rift proved to be quite a small one, yet the patient had succumbed within two or three years. It may well be that in these many cases of quicker dilapidation, as in this one, the

[1] Grau, "Luetische Aortenerkrankungen," *Zeitschr. f. klin. Med.* Bd. lxxii., 1910.

[2] Stadler, *Die Klinik d. syphilitischen Aortenerkrankung*, a valuable monograph, based upon 200 cases and 150 necropsies. Jena, 1912.

coronary orifices were occluded; I have not myself made an adequate comparison, but the surmise seems reasonable. As with other side vessels, the process rarely extends far on the coronary arteries; and if one only be blocked, myocardial function is still maintained by anastomosis (*e.g.* p. 267). In another part of this book (p. 361) I have proved, it is true, how wonderfully a heart may hold its own even with blocked coronaries, but under other conditions: in these cases—cases of atheroma—no cardiac hypertrophy may be required, and probably their mouths are closed very gradually; moreover these changes befall persons of an age when life and its avocations are confined within a narrower and narrower circle. If in the syphilitic cases the orifices be blocked, the myocardium will present evidences of grave degeneration, although within the muscle the coronary branches may be normal throughout their course. The following case from my own notes, in which the coronary arteries were probably blocked, may be cited here:

Male patient of about 35, seen with Dr. M'Ilwaine, then of Redhill, who recognised the nature of the case, and watched it closely throughout. Syphilitic aortitis was plain enough to the instructed observer. Soon the valve began to suffer, and at first with a remarkable intermittency of regurgitation, as if it depended more on relaxation of the orifice than on destruction in the cusps. The reflux murmur, as we both observed, went and came until seven or eight months before his death, when it became persistent. Before this period he improved substantially on antiluetic treatment (mercury, etc.), was able to fish and shoot a little, and to play a quiet game of golf. Then dyspnea set in, his legs grew puffy, and a systolic murmur became audible at the apex also. In spite of all this he decided to sail, with a medical attendant, for warmer seas, and on shipboard to take complete rest. The result was better than we expected, for with this rest and change of air, combined with digitalis and other appropriate means, the mitral murmur vanished, and other symptoms cleared up. But a faint aortic diastolic murmur still persisted. He improved but for a short time; ere long the mitral murmur reappeared, and with it other signs and symptoms of heart failure, not this time to be dispelled.[1] (See case, p. 529.)

In the Hodgson-Welch aorta, however bulging, as in

[1] I have not thought it necessary to insert other cases, although I am somewhat rich in notes of them. They differ only in degree, and I have blended their characters in the text.

aneurysm, the heart is not called upon for overgrowth; for in neither case ordinarily, as we all agree, are the arterial pressures raised. Stadler says that in Hodgson's disease, as in aneurysm, if there be no valvular nor renal complication—which often of course there is—their range is usually normal throughout. He has found no reason to assent to the alleged great frequency of renal disease in these cases;[1] he agrees however, as from the state of the aorta we should expect, that the pulse amplitude is enlarged, the systolic pressure being relatively high and the diastolic low (40-70 mm.).

In syphilitic regurgitation it may be asked if this lack of adequate response on the part of the left ventricle may not be due to the co-operation of a gummous lesion in the heart itself, below the valve? Now it is a curious result, but so it is, that syphilis of the heart and aorta, a combination which one might expect to be frequent, is really somewhat infrequent. The heart substance is attacked, or the aorta is attacked, but it does not often happen that both are invaded together. Thus also, as we have seen (Vol. I. p. 294), the aorta may be gravely infected while the peripheral arteries remain intact. Of course there are many examples to the contrary; especially in those cases in which the infection is otherwise widespread, as in the case I have quoted from Benda, of a woman in the Moabit Hospital, aged only 22, which, besides a syphilitic aorta, and much syphilis elsewhere (including, if I remember right, the portal vein, a vessel sometimes similarly attacked both in acquired and in congenital cases), presented also some syphilitic disease of the myocardium. Dr. Lauriston Shaw (*Lancet*, Sept. 23, 1900), at the necropsy of a man, æt. 26, who died suddenly during apparent health, but with a history of both strain and syphilis, found the aorta beset with "atheromatous" patches, and an aneurysm on the right of the root of the aorta, over the right ventricle. There was a gumma in the heart near the apex, and a small one in the pancreas; in all other respects the heart and the rest of the body were healthy.

The portal vein lesions hitherto described seem to have been rather of the intimal type; more like atheroma with

[1] Leube, Rose Bradford, and others. See chapter on Renal Disease and Arteriosclerosis, Vol. I. p. 367.

break-up of the elastica and degenerated media; thence follow thrombosis and the consecutive visceral evils. The records of Pick, and many other pathologists, contain cases of this combination, though often the clinical notes are not given. But the irregularities of distribution are endless. I remember a terrible case of virulent syphilis, ravaging, among other parts, the larynx, but the heart and vessels, to the best of our opinion, were intact, and the patient made a complete recovery.

The association of this *disease of the aorta with tabes* was first pointed out, I think, by Berger and Rosenbach.[1] But I have noted how frequently in aortic syphilis this lesion exists alone; often indeed without any accessible stigma—unless it be by the Wa.R. test—of the parent infection. My own impression is that the association is more common in tabes than in general paralysis, but it may well be because I have seen more of tabes; palsy cases disappear into asylums, while the tabetic remain in our general hospitals, and come to necropsy there. I find, on comparing many published records (Chiari, Straub,[2] and others), that in tabes it may be no exaggeration to compute that some degree of syphilitic aortitis, if duly searched for, may be found in 40 per cent; possibly in more. And we may surmise that *the proportion in general paralysis* may be no less. Conversely, Goldscheider,[3] in 136 cases of syphilitic aorta, detected 29 of tabes; but the symptoms were often slight, and easy to be overlooked. Deneke[4] reported that of 173 patients with syphilitic aortitis 40 per cent had tabes, and in one-tenth of the rest it was suspected. By the Wa.R. 87 per cent were positive; 6 were dubious, and 16 negative, but of these 13 were under active specific treatment; moreover as the staff became more expert in the method the negative instances were becoming fewer and fewer. In all cases of luetic aortitis, as a matter of routine, the pupils and knee-jerks should be tested.[5]

[1] Berger u. Rosenbach, *Berlin. klin. Wochenschr.*, 1879.

[2] Straub, *Verhandl. d. deutsch. path. Ges.*, 1899.

[3] Goldscheider, *Wien. med. Klinik*, No. 12, 1912; quoted *Epit. Brit. Med. Journ.*, Aug. 10, 1912.

[4] Deneke, at a meeting of the Medical Association of Hamburg in 1912; quoted in the German journals about that date. See also p. 167.

[5] See also Lippmann, a worker on this subject, *Aortenlues*, Festschr d. allg. K'haus, Hamburg, St. Georg, 1912; quoted *Zentralbl. f. Herz- u. Gef.-Kranktn.*, Nov. 1912.

The number of these patients who admitted infection, or indeed seemed to be aware of it, were a small fraction.

Deneke says that the death-rate of the syphilised is 68 per cent above the normal level of insurance computation; well may he denounce the sequels of syphilitic infection as "ungeheure!" Of 83 deaths from syphilis in the Hamburg Hospital during 1909–11, 54 were due to aortitis; the rest to other specific lesions.

To turn now to the *morbid histology* of the process: we are prepared to accept syphilis of the aorta as a peculiar lesion, a lesion which has an origin, characters, and issues different from those of other affections of the aorta: *different from atheroma, with which however in elderly cases it is often mingled*; and different, though less different, from other infections of the vessel. In histological mode the rheumatic aortitis is not unlike it, and like it is often associated with regurgitative lesion.

We have seen that the seat of syphilis in the aorta is curiously definite; that ordinarily, like the rheumatic poison, but with even more constancy, it fastens upon the vessel near to, but a little above, the semilunar valve; or about the mark of the ductus botalli. Welch pointed out this proclivity, and the tendency thence to spread upwards along the transverse arch, and downwards to the valve. The lower margin is but too prone to creep to the mouths of the coronary arteries (p. 265). Often the disease occupies the descending thoracic portion also, and rarely may reach as far as the forks of the gastric and renal arteries; usually it terminates, with a sharp limit, at the diaphragm. In some cases the innominate is involved, but, save that occasionally it blocks their mouths, the disease does not spread far into the side vessels. In Jores' Case VII.[1] the thoracic lesion, though characteristic, extended to the descending aorta and cœliac axis (*vide et* p. 309).

With other infections the syphilitic has these characters in common, that it seems to invade the vessel by way of its *vasa vasorum*, and to be, as Heller[2] and Chiari showed, an inflammation; that is, an irritative lesion with reparative

[1] Jores on p. 382 of the *Virchow's Arch.*, 1904, paper. See also Case VIII.
[2] Heller, A., *Münch. med. Wochenschr.* No. 50, 1899.

reaction ; as contrasted with the degradation of atheroma, which is not directly due to a specific infection. A cellular hyperplasia, with the exudation of a hyaline substance, follows the courses of these vessels, many of which become occluded. Thus by the vasa vasorum lues is instilled into the aortic wall ; but accordingly its chief activity lies at first more *in the adventitia*, where this network is abundant, and between this coat and the media. The adventitia may become thickened by several millimetres. The media, as in the rheumatic kind, soon follows, and with perilous consequences ; but the intima, rarely the seat of a primary lesion, suffers last, though it is practically always thickened by fibrosis, and often exhibits a patchy proliferation of the subendothelial tissue. About the vasa vasorum and the adventitia the proliferative features resemble those which Dr. Mott produced by painting the outside of a vessel with an irritant solution, or those of vessels involved in contiguous inflammations, or again of certain experimental infections with bacteria. In an early phase then the vasa vasorum are the active seat of cell proliferation; like active changes become patent in the outer media, and, directly or obliquely, pass more deeply into this coat, so weakening it that it becomes less and less able to withstand ordinary blood pressures. Some observers, Molinari [1] for example, believe that the outer elastica suffers early ; in the first instance its injury may be due to the buckling of the wall. These changes in the adventitia are often coarse, and may not bear upon the face of them any syphilitic stigma, unless here and there they form masses which may be called gumma (Benda). About such fresh foci, in the external media and adjacent layers, the specific spirochæta has occasionally been found (Reuter,[2] Benda, Schmorl and others), on the whole perhaps not infrequently ; so that the question whether this aortitis be "parasyphilitic," and the exudative elements non-specific, has fallen to the ground. Gruber quotes Vanzetti [3] as having produced a characteristic aortitis in animals by direct infection with the spirochæta.

[1] Molinari, G. (of Breslau), *Schwielige Arteriosklerose u. Syphilis*, Leipzig, 1904. A Dissertation for Doctorate.

[2] Reuter, *Zeitschr. f. Infektionskhtn.* Bd. liv.

[3] Vanzetti, *Arch. p. le sc. med.* 35, No. 24.

My own impression is that definite gumma, such as we find in heart or liver, is in the syphilitic aorta rare (Vol. I. p. 300). This name can scarcely be given to the cushions on the internal aspect, characteristic and salient as they are, unless it be on the ground of healing by scar. Kraus would make two classes, of gummous and diffuse aortitis; but the difference is superficial; "gumma" is itself not histologically peculiar, but a concentrated focus of proliferating fixed cells and wandering cells. Whether called gumma or not, the phase may be ranged in the direct line of infection. In case of doubt, all other parts — eyes, skin, testicles, etc.—will be scrutinised for specific inscriptions. One small circular scar on leg or arm, with sharp walls and white and silky floor, may suffice to clinch the diagnosis [1] (see p. 210).

The aortic bulges, infiltrated and thickened as they are, probably owe much of their outward protrusion to the yielding of the media; though a diffuse toxic necrosis of the muscular and other structures, plain enough in sections of the media, is of course not peculiar to lues; it is well marked also in the rheumatic kind. In the adventitia these collections of plasma and lymphoid cells, around the vasa vasorum or radiating from them, are often disposed to an annular distribution, as we see in the larger cushions visible to the eye. It is said (Fukushi [2]) that new growths of elastic, as well as of connective (*kollagene*) fibre, may be seen in the adventitia. But of course in the necrotic portions the elastic fibres suffer early, lose stain, and break up; a perishing which may be secondary to that of the muscular coat. Fukushi thinks that the plasma cells and lymphocytes are so abundant as to give a specific character to the exudation; but I agree with Klotz [3] that similar phases may be seen in rheumatic and other kinds of aortitis.

In the media we see lesion both spreading inwards from the adventitia and established also in separate foci; nests of plasma cells attached, as it were, to the finest inward twigs of a vas vasis. These vessels, after the first congestive and proliferative phase, silt up, and thus entail the atrophic phenomena which ensue upon the inflammatory; and this more and more as time

[1] See Appendix, p. 210.

[2] Fukushi (of the Berlin University), *Virchow's Arch.* Bd. ccxi., 1913.

[3] Klotz, O., "Rheumatic Arterial Lesions," *Journ. Path. and Bact.* vol. xviii. No. 2, Oct. 1913.

goes on. There are nevertheless, as we shall see, large areas of healing by scar. Between these foci of active mischief may be seen, both microscopically and by the naked eye, areas of quite normal structure ; although, as the process extends, these normal areas gradually merge in the universal degradation. This "mesaortitis productiva" [1] is very severe in the neighbourhood of an aneurysm. The miliary foci in the media, sometimes called, as we have seen, a little figuratively, "miliary gumma," may be, indeed usually are, intensive and extensive ; so that (passing by saccular aneurysm, with which I am not concerned) the media buckles outwards more or less, here and there, according to the inequalities of the distribution of the disease ; but (aneurysm apart) these bulges are usually basin-shaped and contain no clot, or but a pellicle. Sections of the wall show all these processes very clearly, as in two specimens kindly sent to us by Dr. James Mackenzie, and prepared, mounted, and drawn in colour under Professor Woodhead's supervision.

The intima, in contrast with atherosclerosis, if not intact, is ordinarily, besides any injury by dislocation, not much the worse, sometimes possibly a little the better ; for it may be thickened, even rather densely thickened, so as to line the cavity of a hollow, or part of the cavity even of a saccular aneurysm ; a fibrotic thickening which may have some protective value. But occasionally, as in certain specimens which Professor Teacher showed me recently at Glasgow, the intima may itself also become the seat of proliferative foci quite detached from those in the media (Vol. I. pp. 302 and 479) ; and about the intima there may be a good deal of diffused proliferative activity of the simpler kind.

In the later phases of the luetic process, so long as atheroma does not supervene, appears, as in inflammatory lesions, a strong tendency to scarring, to a kind of repair by fibrosis,[2] as contrasted with the fatty and calcareous perishing of atherosclerosis. From the adventitia inwards, and also in the media and subintima, so widespread a fibrosis goes forward that while at one or more spots active granulomatous change, if such it be, is still in progress, at older spots scars are forming, with

[1] This name, given by Chiari, fails to connote primary attacks on the adventitia.

[2] See Wiesner, *Centralbl. f. allg. Path. u. path. Anat.*, Jena, 1905 ; Klotz, O., *Journ. Path. and Bact.*, Oct. 1907, and some ephemeral papers of my own.

puckerings, pits, and pouches; some parts scooped out even to translucency; various stages of disease which give to the Hodgson-Welch aorta its characteristic aspect. Benda says that some of the smaller patches may form scar and heal without deformity. It is true that in a small way scar may appear in atherosclerosis also, but quite subordinately, not characteristically. Indeed apart from acute infections scar is rarely discovered.

The process of *extension to the aortic valve* has been minutely examined by Dr. Sidney Martin.[1] The vessels in the valve segments begin to thicken; in the cusps, and beneath them, a general periarteritis is set up after the fashion peculiar to syphilis. Thus the valvular membranes themselves thicken, scar, and pucker. The same process may be seen in the sinuses, so that here again the machine may be much deformed; but, if taken early, the mischief may perhaps by specific treatment be dispersed. I may add that, more commonly in syphilis than in atherosclerosis, the attachment of a cusp to the wall breaks away, throwing two limbs of the valve together.

The *pulmonary artery* may be attacked in like manner, but is far less liable to the disease than the aorta; usually it escapes, but not always.[2]

I repeat that these changes and their seat give to the Hodgson-Welch aorta within and without a peculiar aspect,[3] which, in cases under middle life and uncomplicated with atherosclerosis, cannot be mistaken. At Leipzig, in 1912, Professor Marchand spread out before me ten of these aortas, so recent that they had not yet been put up in their jars. About no one of these, to the practised eye, could there have been the slightest doubt. Specimens may be found in almost any good museum. The arch lies before the observer widely dilated, distorted, thickened, pitted, pouched, puckered and scarred out of all recognition; indeed I have known such a specimen taken at first sight for an ulcerated colon. In parts, as I have said, the wasted and cicatrised wall may become so thin and pouched as to be translucent.

In the earlier stages of the aortic infection the vessel is not

[1] Martin, S., *Clin. Journ.*, June 20, 1907.
[2] See Barth, H., *Frankf. Z. f. Path.*, 1910.
[3] See illustration (coloured) in Balfour's *Dis. of the Heart*, 3rd edition.

so much dilated and deformed ; if viewed from without, perhaps scarcely at all. It is on the inner face that the more or less annular, raised, cushion-like tumidity may be seen, lying a finger's-breadth, or a little less or more, above the valve. It is in my experience pink, at any rate at first, and except for its saliency, and a certain succulence or translucency, scarcely visible above the surrounding mucous membrane. But on the surface of the syphilitic cushion a rough granular appearance may be perceived (p. 262), and, when recent, its boundaries are fairly sharp. The parts from a case of incipient aortitis which Dr. Parkes Weber brought to my lecture on Angina Pectoris at Fitzroy Square in 1908 had these characters (p. 264). Such also was the form of Brault's case (quoted by Letulle) already referred to. In other and probably riper cases the wheal is said to be yellow, or yellowish white, but to differ in form and colour from the more opaque atheroma. Tripier [1] regards all gelatiniform patches, whether associated with atheroma or not, as syphilitic. I am disposed to say that, in translucent greyish or even pinkish tint, influenzal or rheumatic patches, or even the acuter extensions of atheroma, may have a like aspect. The criterion is the lack of fatty and calcareous change, so long, that is, as atheroma may not have supervened, a mosaic often to be seen in later stages and later years. Besides these larger cushions, smaller ones also, like peas or seeds, may be noted here and there upon an otherwise normal inner surface ; these are apt to join together so as to form the padded ring I have described. As I shall point out in the essay on Angina Pectoris, it is of great importance in early cases to inspect the vessel carefully from the outside, as the adventitia and the periadventitial lymph-glands may be thickened and dense with infiltration, but at a glance show little that is morbid. Yet in a square centimetre, or less, of this fibrous sheath may lie the long agony of angina pectoris.

Now how in life are we to detect this insidious but deadly evil in time to arrest it before these vital parts, especially the mouth and valve of the aorta, are destroyed ? If a patient is known to be syphilitic, a close watch over this among other vulnerable parts may enable us to infer its presence, and to scotch it before too much harm is done ; the supreme difficulty is with

[1] Tripier, *Études anat. clin.*, Paris, 1909.

persons not recognised as syphilitic, and in whom no characteristic sign or symptom had arisen until the lesion of the aorta had made some considerable way. Now that the spirochæta is found in tabes and general paralysis the parasyphilitic distinction will probably vanish; the question will be whether or no the specific remedies can be directed to the part diseased. That the spirochæta is not readily found in aortitis, and not yet in the media, is no great matter; it is likewise scarce in periosteal gumma. We have seen indeed that it is not easy to pronounce upon the form of these hyperplastic deposits, as definitely granuloma; they may present no such criterion of specific origin, but they are quite consistent with "tertiary" syphilitic outbreaks elsewhere. My own opinion and experience tell me that these aortic outbreaks are "tertiary," and in their degrees amenable to specific medication, whether by mercury or salvarsan. They are not, I think, very amenable to the iodides; more than once after large and long and ineffectual dosing with these salts in vain, I have seen the symptoms and signs of aortic lues recede during a subsequent course of mercurial inunction. There is room for hope then that, if we can but detect the disease early, we may arrest it before it does its worst.

That in some cases of aortitis we have to deal with *mixed infections* is not improbable; but on this point we have little definite information.

Of acute aortitis as a result of **lead or other mineral or vegetable poisons**, I have but scanty notes; but I may state, not from my own experience only, that in some plumbic cases aortic disease may appear without renal disease. Dr. Seymour Taylor[1] has published a case of plumbism with anginous symptoms, followed by those of ruptured aortic valve.

On **muscular stress** as a cause of aortitis in a vessel otherwise sound, I have little to add to what I have written under Atherosclerosis (Vol. I. p. 205). That such stresses co-operate with more insidious causes of aortitis—acute or chronic—I have said already. The far greater prevalence of the Hodgson-Welch aorta in men suggests some co-operation of bodily labour.[2]

[1] Taylor, S., *Lancet*, Feb. 22, 1908.

[2] See *e.g.* Grassmann, *Münch. med. Wochenschr.*, 1907; and Hesse, *Arch. f. klin. Med* Bd. lxxxix., 1907.

I recall one case only in which mechanical stress seemed to have originated a definite aortitis; and in this case the strain may have co-operated with some unrecognised infection. Later enquiries indeed suggested a rheumatic history. The patient was a young housemaid who, on lifting a bedstead, a weight beyond her strength, felt a sudden severe pain and distress in the chest, which persisted in great severity. She came several times into the Addenbrooke's Hospital, when Dr. Lloyd Jones called my attention to the case; and she was once or twice in St. Thomas's, when Dr. Hawkins kindly sent me news of her. The signs and symptoms were those of acute aortitis, subsequently becoming chronic and invading the aortic area so far as to cause stenosis. Her crippled state, and the angina pectoris which continually beset her, preyed upon her spirits, and after a while engendered, not unnaturally, a fretful and wayward disposition, so that she was accused of hysteria and of mock angina; but on the main facts of the organic lesion we were all agreed. The angina was typical and very severe. After long suffering this young woman quite recovered her health; but a harsh systolic aortic murmur and a thrill, at the base, present throughout the illness, still bear their permanent witness.

Of other agents in the causation of chronic aortitis, I may touch so far again upon *gout* (Vol. I. p. 275) as to say that chemical research fails to discover urate of soda in the aorta of gouty bodies; and that experimental injections of uric acid have no effect upon the vessel: therefore the attribution of the disease to silting up of the vasa vasis by uratic crystals is likely to share the fate of many another ingenious guess.

On **the experimental side of the subject** definite results are difficult to attain; experiment on the generation of aortitis by bacterial agency is obviously intricate, and in its results equivocal. Nevertheless, some instructive work has been done. Gilbert and Lion's successful experiments in 1888 in producing an infective aortitis by injections of bacteria and their toxins are well known (Vol. I. p. 285). The pneumococcus, the B. coli, Eberth's bacillus, and pyogenic organisms, have been inoculated upon the aorta in animals with no inconsiderable success; a success now sufficient to indicate to us that such microbes are capable of engendering acute aortitis, if circumstances be favourable to

their implantation. See Jordan's case (p. 161). The case of the horse poisoned by diphtheritic toxin (p. 164) seems to prove, moreover, that in the absence of the microbe itself toxins of bacterial origin may suffice to produce this inflammation. I have already suggested that it may be more easy for toxins than for bacteria to fasten upon a large artery.

As we pass from the acuter to more **chronic modes of aortitis** we are still beset by the obscurity and incertitude which cast their shadow over this clinical field. Thus, disease of the ascending aorta, as distinguished from diffuse atheroma or arteriosclerosis, receives less attention than its due; it may occur alone, or nearly alone, and, if its symptoms be often ambiguous or equivocal, it is quite capable of compassing the patient's death; so that we shall do well to suspect it, and to interrogate the signs of it more warily. On two recent occasions I ventured, and ventured as it proved with justice, to persist in a diagnosis of lesion of the ascending aorta against the judgment of colleagues who did not so much deny it as ignore it. Chronic disease of the ascending aorta, without aneurysm, is far more common in middle life, in early adult life, and even in youthful life, than we are wont to realise, and is for the most part insidious. On the signs and symptoms by which it is to be known I have said much and shall say more in the essay on Angina Pectoris. When the patches are small and few, the evidence, if any, may be wholly indirect. Let me repeat that the name atheroma is conveniently restricted to the decaying—the necrotic—phases, which may so appear from the beginning, or may follow inflammatory processes as indicated by the name aortitis. When inflammation or, in the particular case, repair, is predominant, we speak of the process as aortitis; when intimal degeneration prevails we speak of it as atheroma. But in the course of chronic aortitis and atheroma—as in the course perhaps of all chronic maladies—tides of acute activity may, and often do, take place; and the uniform course of the patient's symptoms may be disturbed by vacillations and exacerbations. Thus it is that symptoms of angina pectoris may manifest themselves in patients in whom the physician had recognised no more than a gradual and less dreadful mode of decline.

In high blood pressure with or without renal disease we are all agreed that the higher the pressures the more the arch of the aorta will suffer; and that, because it bears the first and chief brunt of the cardiac systole, the first portion of the ascending limb of the arch, is the first and chief seat of atheroma. The surprising thing is that the effects are not sooner manifest (Vol. I. p. 190). Earlier or later however, in every case of static hypertrophy of the heart, the aortic tissue deteriorates. The vessel suffers from the impact geometrically more and more as the blood pressure rises, and the vessel with age loses its resilience. As we have seen, in the chapter on Physics, if resilience fails, normal and indeed subnormal blood pressures become relatively excessive; expansion becomes thumping, the machine is self-strained, and, as this and that part of the vessel yield unequally, stresses become unequal. We have seen that as a pipe becomes rigid, its movement is more abrupt; especially at a bend, such as the aortic arch, which at each beat is hammered tangentially forward as a whole. Dilatation, in such a state of the vessel, may possibly bring with it some slight compensation in a reduction of velocity. Yet, how damaging the heavy pulses often are, we may read in the immunity of the arch when it is protected by aortic stenosis of the so-called pure kind —the kind in which a big heart and a straitened aortic ring exist without notable disease above the ring; in these cases, as in two of my own, the aorta, although in old age, may present little or no sign of decay. Where, on the contrary, as in Hodgson's disease, the heart is unimpaired, the aortic orifice open, and the vessel itself softened, we see degradation and deformation carried to the extreme verge of survival.

Symptoms and Diagnosis.—As the syphilitic is by far the most common and the most grievous of the kinds of aortitis it is more convenient and concise to begin with a description of the symptoms of aortitis as observed in this kind. Thus we shall best learn the clinical phases of aortitis and, by no difficult comparison and inference, the behaviour of the other kinds. We have seen that, heavy as is the incidence of aortic syphilis, in not a few cases — after the manner of the disease — the aortic outbreak

stands either alone in the arterial tree (Heller and Döhle, and others) or with arterial disease in inaccessible parts only, as in the heart or brain. Soft peripheral arteries are no warrant against disease in the aorta or heart. Gruber says that "general arteriosclerosis" comes on in the later stages of the disease; but we have seen that this result is not invariable, nor even general (Vol. I. p. 295). We know too well in how many cases of syphilitic disease the primary infection is unknown, and the patient himself taken wholly by surprise. But, as in tabes so in syphilis of the aorta, it seems needful to reiterate that, in cases of which the specific origin is beyond doubt, the absence of a history of infection is a common experience, and should cause no surprise to the physician. Here the serum reaction is invaluable, though of course a positive Wa.R. does not prove every associated affection to be luetic. A man aware of a past infection should be wise enough to submit himself to a thorough examination at intervals during the rest of his life, or at any rate up to fifty years of age; especially on the occurrence of any sort of symptom of ill-health or frailty. In a subject of tabes or general paralysis aortitis may almost be anticipated.

Of *symptomatic fever* it is hard to find definite and specific records. Moreover this symptom may be merged in the fever of a parent disease. Still, as in Dr. Windsor's case (p. 157), the febrile curves, though not of large excursion, may be distinct enough. Popoff[1] is quite clear that aortitis, like endocarditis, has its fever—various perhaps as its specific cause. He has noted it, as I have done, in the syphilitic kind.

That *pain*, and pain having peculiar and even alarming features, arises in disease of the aortic area—that is, of the first portion of the ascending aorta and perhaps of the aortic orifice—is in a sense well known; in another sense it is ignored or misconceived. Such is the tenacity of opinion, such are the fetters of fixed doctrine upon plain thinking, that, as I shall say in the essay on Angina, in the examination of cases of angina pectoris our eyes have been too exclusively fixed upon the coronary arteries; the orifices and the tunics of these vessels are scanned for atheroma, and if atheroma be found—in chronic disease of this area they hardly escape some measure of it—the

[1] Popoff, *Zeitschr. f. klin. Med.* Bd. lxxv., 1912.

pain is held to be explained, and no fresh thought is given to the matter. Yet the truth is that this pain, which in greater or less degree is a predominant feature in disease of this area, is due, nearly always neither to coronary artery nor to heart, but to disease of the aorta, with which coronary atheroma is commonly associated. Now it is true that a superficial internal aortitis of the arch, even when acute, is unattended with pain, or, if some pain be complained of, it receives little attention ; in the aortitis of the infections pain is frequently, in some of them generally, absent. In the aortitis of smallpox, or of typhoid fever, we detect the evil less by pain than by physical signs, by later consequences, or after death. In the aortitis of influenza, or of rheumatism, pain arises in a minority of cases ; in syphilis it may be more frequent. In atheroma of this area the cases of pain are a small minority. There is no direct relation then between severity of pain and the cause, the acuteness, or the superficial range of aortitis, or atheroma ; the intensity of the pain in certain cases is explicable, as I shall suggest, by the depth of its penetration or thrust towards the outer investments of the vessel. In certain circumstances it is admitted that, whatsoever pain at the heart may be, pain having peculiar and even appalling features may arise in disease of the aorta, especially of the suprasigmoid portion which seems more obnoxious to infective attacks. Its characters are : origin under the sternum, often at the junction of the first and second thirds of the bone, and sense of constriction or compression, as if the chest were in a vice, or the breast-bone crushed inwards by an iron bar. And this pain may come in any degree, from a transient sense of tightness or oppression about the upper sternum, to utter torture. A third feature is a tendency to radiate from the mid-sternal origin into sections of the brachial plexus, in the first instance, of the left side, but occasionally of the right also—rarely of the right alone. The zones of this aortic pain may be divided into the primitive zone, and the forced zones ; the primitive zone being that of simple reference, the forced areas those of compound reference ; these are invaded successively as the irritation, or the instability of the corresponding spinal and other segments, increases. As in a child a cutting tooth may have a primitive area of pain in the parts about it,

and thence may force sphere after sphere of inhibition, to vomiting and even to general convulsions, so a corrosive lesion at the first part of the aorta may cause a constricting pain under the sternum only, or, forcing successive spheres of inhibition, may drive to the shoulder, to the inner condyle of the humerus, to the ring and little finger, to the neck, and so on. The pain of a diseased aorta—angina pectoris in fact—having invaded the outer and inner bailey, may force the very keep of life, and arrest the heart in fatal syncope. Herein, as we shall see, lies the peril of aortic pain to him whose heart is frail; to him, let us say, whose coronary arteries are occluded, or—which by the way is far from being an equal consequence—whose cardiac muscle is in decay. To one thus unstable at the centre angina pectoris may be a mortal agony; to one whose heart is sound and perfect, or relatively sound and perfect, the agony, if no less intense, is rarely mortal. In such cases the heart may not quail, and the sense and apprehension of death, if present at all, are less terrible, or indeed absent. Yet notwithstanding, in acute aortitis, as in other agonising visceral pains, even a sound heart may be arrested in death.

When a chronic aortitis, pursuing for the greater part a painless course, is broken by terms of acuter activity, its own pain may come and go with such vicissitudes; as I shall show in the essay on Angina (p. 426). I have seen many such cases of intercurrent aortic pain, in acuter phases of chronic disease, which departed for good. In acute aortitis its departure is perhaps the rule, on condition that the heart is not degenerate. Stadler's admission (*loc. cit.*) that incessant anginous attacks may be aortic, but periodical attacks coronary, cannot withstand a moment's consideration, the difference is but one of degree; in the same patient we may find all degrees, from slight "stenocardia" to the "status anginosus." I suggest that this symptomatic climax depends directly not upon the acuteness of an aortitis but, as I shall explain more fully under Angina Pectoris, upon its penetration, on the degree of distension of the capsule of the vessel, or of drag upon it. Dread is a criterion between aortic and coronary disease only thus far—that a young and vigorous heart often outbraves this omen, which seems more frequent in old persons, or those whose heart is frail. However it

is no infrequent feature of intense aortitis of infectious origin in persons under middle age. For instance, in the remarkable case described by Rist and Krantz[1] of syphilitic aorta in a patient æt. 37, when the angina was continued and severe, the attacks were attended with *sensation de mort imminente*. Yet, as regards their mouths, trunks and intramyocardial twigs, the coronary arteries were intact. "*Il y avait donc aortite sans coronarite*"; but the symptoms were angina pectoris, typical and complete! The kidneys were normal. The patient died of pulmonary œdema. And in typical angina, of elderly persons, we shall see that dread is an inconstant symptom; it is often less manifest in poignant cases than in attacks which otherwise, however sinister, are less painful. If in aortic disease, as I have suggested, and Bittorf also, the vagus terminals may be deleted, in this chance we may find some explanation of absences or abatements of the dread. These remarks on aortic pain seem proper to this context, but I must not trespass too far on the essay devoted to Angina Pectoris, where the nature of such pain will be closely debated. Then also I shall discuss the inconstant symptoms of local tactile tenderness and sensibility on the chest, as described by Peter,[2] and by others later.

If then the inflammation happen to light upon the investment of the aorta in such a way, or at such a rate of activity, as to make the vessel abnormally sensitive to the pressure of the output of blood, so sensitive as to propagate the stimulus to the sensorium, a "stenocardial" sensation is an early symptom (see essay on Angina Pectoris, p. 287). It is remarkable that Deneke is almost alone in not mentioning slight angina ("stenocardia") as an early symptom of syphilitic aortitis.

Again, the first betrayal, if perchance it be looked for, may be by some modification of the X-ray picture; some yielding of the vessel, some distortion of its contour, some darkening of its shadow suggestive of thickening (p. 204).

Stadler says that among the early, or more indefinite, symptoms may be a sense of *general fatigue* (*scheinbar neurasthe-*

[1] Rist et Krantz, *B. et M. de la Soc. Méd. des Hôp.*, juin 28, 1906.

[2] Peter, "Exploration de la sensibilité locale des régions précordiales et préaortiques dans les maladies du cœur," *France médicale*, 1881.

nische Initialsymptome); he suggests that this debility may be an indication of closure of the coronary mouths, but this is probably a later extension. In some cases of my own in which such fatigue was not noticed, or not until the mischief was very far advanced, these vessels may have lain longer open to the tides of the blood. I have remarked this fatigue especially in the cases of aortitis with regurgitation, a manifestation for which there may be more than one reason; in these cases the coronaries are often occluded. Again, a specific aortitis, working round the root of the aorta, may invade the conducting tracts, as rheumatism or atheroma may do, and set up the phenomena of heart block—this in one very interesting case I saw and followed out. In this site periaortitis may give rise to a friction sound. The *earthy complexion* and *falling hair* of the syphilitic patient, in contrast with the rheumatic type, may be notable. The *pulse* may be unaffected, or simply febrile, or, in acute cases, bounding with the throbbing aorta; in severe cases it may intermit (vagus). In Morison's case (*loc. cit.*) the vagus on one side was involved in the specific process. The subclavian artery may be raised, vibrating, and hammering (Vol. I. p. 395). *Pressure symptoms* are, of course, late phenomena of chronic cases; the earliest I have noted is the rather strident "whistling" respiration on exertion which, though by no means an initial sign, may yet long precede other respiratory signs, and the gravest issues of the malady. This symptom I remember especially in two syphilitic cases, neither with aortic regurgitation. The whistling is not distressing to the patient, at any rate not at first; it is often more perceptible to the bystander than to the patient himself. In two of my own cases spasm of the glottis was so intense as to call for chloroform; and in one of them for tracheotomy. The following case is interesting in this respect:

A man, æt. 44, found his neck enlarging, his face and chest puffy, and breath short. His physician found some blueness and slight œdema of his arms and dilated superficial veins about the neck and chest. Yet aneurysm of the aorta or mediastinal tumor seemed improbable. By X-rays, however, we detected dilatation of the ascending portion and first part of the arch of the aorta, which was pressing upon the upper vena cava, the right bronchus, and the left

recurrent nerve. Syphilis was strongly denied, but a specific iridochoroiditis banished all doubt. Under mercury and iodide of potassium this patient made a complete recovery.

Stadler emphasises an early and insidious and more disturbing dyspnea, "not obstructive but functional"; in my experience this has not been an early symptom, but one rather of the stage of regurgitation, or of incipient heart failure.

We may now concentrate our attention upon *three cardinal methods of the physical diagnosis of aortitis*; that is, upon the percussion, auscultation, and ray methods. Of the first two methods I may speak with personal conviction, for my experience of them is not inconsiderable; of the last my knowledge is based upon the demonstrations and interpretations of expert colleagues. However, I have found these testimonies, in cases of which I have had personal knowledge, consistent with published records, and with each other.

The normal aorta of a young adult man should measure 5-7 cm.; over æt. 50, 8 cm. In women it is rather less.[1] It is difficult to say at what lapse of time after the initiation of an aortitis the vessel is enlarged in both diameters—in width and in length—and how much may be attributed to atony. It may be that the increase in length, by tortuous displacement, does more than the increase in width to thrust the vessel towards the chest wall, and so to *damp the vibrations of the sternum and nearer parts of the right ribs.* At first sight it would seem that such a displacement would not come about until the vessel were gravely affected; but, as atony may have some part in the expansion, this alteration need not run parallel in degree with the lesion. Stadler (*loc. cit.*) has some interesting remarks on the atonic and the destructive dilatation of the aorta in syphilitic cases; and again on confinement of the vessel to narrower limits by rigidity or adhesion. These are points on which post-mortem evidence can be only indirect; some evidence however there is—or so it is said—of a recession of dilatation. For my part, in syphilis I have no recollection of this *area of dulness*, once established, contracting in any appreciable

[1] See study of relative diameters and areas in man and animals by Dreyer, Ray, and Ainley Walker, with calculations of sectional area from body surface (*Proc. Royal Soc.*, Dec. 1912).

degree; nor have I succeeded by the methods of Abrams[1] or Ewart (percussion on the seventh cervical spine, etc.) in bringing about the slightest reduction of its compass. Indeed in several cases of great alleviation under treatment I have found the area of dulness still to abide unchanged. In rheumatic and other aortitis on the contrary such recession generally comes about. Ordinary atherosclerosis rarely widens the aorta so remarkably. And as, unless in exceptional cases, the blood pressures are not increased, we are not puzzled by fluctuations in the volumes of the heart's chambers. Nor again do the positions of respiration alter the percussion area of the syphilitic aorta. Of a muffling effect on the manubrial fremitus of adhesion around the ascending aorta I can find no note, neither in cases of my own nor in those of others; but the sign is worth looking for. It is said that a corresponding area of dulness may be detected at the 3rd-4th dorsal spines.

On the site of the area of dulness I need not dwell minutely; Potain, whom we have to thank for its explicit demarcation (*loc. cit.*), and many later writers have described it as the dulness "*en casque*"—in the form of a fireman's helmet. If from without inwards, on many radii, the boundaries of it be obtained by careful percussion, the area will be found to occupy the manubrium, or at first perhaps the middle third of it, with an adjacent area of the second rib and third interspace, one to two finger-breadths to the right. In one of my cases (lues) it extended fully 4 cm. to the right, and the outline may slope up from the 4th cartilage. The dulness about mid-manubrium is generally quite decisive, at the top a little less. It may cross over a little to the left side, but is never so dull there as on the right. Of the alleged corresponding dulness in the area of the 3-4 dorsal vertebræ I can say nothing. Dr. Sansom advised the use of the pleximeter for this mapping; the pleximeter is useful for class teaching, but, to me at any rate, the help of the sensibility of the under finger is indispensable. This percussion sign, even if we cannot call it a very early one, I have found very trustworthy.

In the normal person, unless we include spare old persons with deteriorated vessels as relatively normal, the arch of the

[1] See *Brit. Med. Journ.*, July 8, 1911.

aorta is not to be felt in *the jugular notch.* Normally, it lies 2-2½ cm. below; over it lie fat, and layers of connective tissue. It may be palpated in persons wasted, or neurotic with atonic vessels, in the subjects of Graves' disease for example, or when, as in high pressure cases, the heart is hypertrophied. But, as Wenkebach has demonstrated, before deciding the value of this upper palpation the position of the diaphragm must be carefully ascertained. Yet even in the syphilitic aorta the vessel is not always palpable, Stadler adduces two cases of the exception; but, as in syphilis the vessel begins to yield near its root, when this jugular sign occurs it is usually at a stage of the aortic disease when we have other and surer evidence. Moreover it is often palpable in ordinary atherosclerosis, when the subclavian and upper arch are crooked and thickened. In some individuals none of these conditions, however inveterate, seems to produce it; though radial pulse differences, due to straitened mouth, may be present. In the younger subjects of rheumatic aortitis, with narrow jugulum, the vessel may not be, often is not, accessible to the finger; in this disease, unless by atony, it is not so much enlarged. Concerning influenzal aortitis in this respect I have no note. In acute cases I have often found the vessel tender to the touch (p. 427), a tenderness to be distinguished from any discomfort of the palpation.

In this context I must allude again to *the subclavian sign* of Faure (*loc. cit.* p. 349), Gerhardt, von Schrötter [1] and others; but in syphilitic aortitis at any rate, it is not of more than occasional service. When the aorta is stretched and twisted, the subclavian artery, as the patient depresses his right shoulder, may be thrown up in the supraclavicular notch (Vol. I. p. 395).

Some evidence of elongation of the ascending aorta, better than may be afforded by the subclavians, was pointed out by Traube,[2] and verified by Edgren (*loc. cit.*), myself, and others; namely, *mobility of the heart's apex* (Vol. I. p. 396). Undue and even extreme mobility of the heart is seen in other maladies, as in neurasthenia; but by their proper features this and such atonies can be excluded. As the patient turns from the supine position to his left side, the heart's apex will swing outwards,

[1] Von Schrötter, "Erkgn. d. Gefässe," Nothnagel's *Path. u. Ther.* vol. xv.
[2] Traube, *Gesamte. Beiträge,* Bd. iii.

until its beat may shift far outside its normal limits. From its normal place, the heart's apex may fall away even to the anterior axillary line. At the same time the left line of absolute dulness moves to the left, though not to the same degree. We may be satisfied of undue mobility even in an enlarged heart, and by position tests we may be enabled to attribute some extrusion of the apex to elongation of the aorta rather than to ventricular hypertrophy. Points so nice as these, if often useful in particular cases, cannot be reduced to rule; but we may remember that destructive dilatation of the arch, with loss of its "bellows" function, might, by degrees of dyspnea or even by pulsus alternans, simulate heart failure (?). As in the case of aneurysm, cardiac hypertrophy has no necessary association with aortitis, acute or chronic.

If we turn to *auscultation*, we shall find another sign of value, one which should give us an earlier clue; unfortunately it is incapable—as yet—of objective demonstration; as yet it exists only as an apprehension of the mind of the listener, and is inconstant. Whether or no in future inscription of cardiac sounds may represent it we cannot say. The sign is a certain quality of the second sound, that which Potain described as the *bruit de tabourka*,[1] a phrase I have used in teaching for many years, both in Leeds and in Cambridge, because the customary English term, "clanging second sound," is not free from objection. Clang is the rather unhappy term of English acousticians to signify what the French call *timbre*, the quality by which, for example, a certain note on the violin differs from the same on a horn or other instrument. Thus the "clang" of the normal second aortic sound would be contrasted, let us say, with an altered clang in aortitis, especially in the syphilitic kind; for this peculiar quality some other term, such as "tympanic second sound," would be better. *Tabourka sound* would do, should this term become sufficiently familiar to convey the idea. "Clang" is of course a quality other than accentuation or pitch of sound.

However, names aside, the maxim itself is one of no little importance, perhaps of the first importance; of the first

[1] The tabourka, an Algerian drum, made of an earthen pot with a skin stretched over its mouth.

importance if it prove to be peculiar to syphilitic aortitis, and, moreover, if not masked by some accident, as by emphysema, a constant. No less is alleged for it already, but, if I rightly remember, Potain did not regard it as pertaining exclusively to syphilis. Whether or no it be a constant is another question; the timbre of a sound is not always identical day by day; in the same person it wanes, waxes, or changes. Unfortunately, as I have said, this criterion is a subjective one, one, so far as I know, not as yet reduced to record; though by acoustic delineation it may hereafter be discerned. Personally, I am not yet prepared to say that this tympanic second, as contrasted with other kinds of aortitis, or even with atherosclerosis, pertains exclusively to syphilis; nor again that in syphilitic aortitis that it is a constant. In incipient Bright's disease I have heard second sounds hard to discriminate from it; but I quite agree that it is characteristic of this aortitis, and that the sign is a useful addition to our means of diagnosis of the disease. I am disposed to explain it by the muffling of the walls of the ascending aorta. In atherosclerosis it does not occur ordinarily, perhaps because this lesion moves rather towards stenosis than towards valvular tension. If the quality may be reinforced by high aortic pressure, it is not by any means dependent upon it. On regurgitation signs I may venture on a word of warning; the murmur may be so faint, so distant, and even so inconstant (see my case with Dr. McIlvaine), that unless, while the patient holds his breath, all possible areas, particularly to the left of the sternum, are traversed, it may escape the physician not well on his guard. That by recurrent fluid veins within the largely dilated arch itself a murmur may be caused during cardiac diastole, I can neither affirm nor deny; if such a murmur may be dispelled, in luetic cases, by specific treatment, and not return, this effect I should attribute to some repair of the valve, or of its seat.

On the signs of regurgitation, and of heart's dimensions and work, I need not dwell; but Stadler says that in syphilitic valvulitis the systolic murmur may by some considerable interval precede the regurgitant; and suggests further that to an expert ear this, sharper, systolic sound may be distinguishable from the systolic murmur of the aortitis. He adds that the peculiar clanging second sound also may continue with its own quality

after regurgitation is established. With the first distinction I am disposed to agree; and this interval may give us time to push forward treatment to prevent the graver mischief: as regards the second I regret that I have made no close observation; on such refinements we need more clinical verification. Possibly Dr. Eustace Smith's "retraction murmur" might be elicited. I may remind the reader that a useful, if not constant, point of distinction between rheumatic and early syphilitic aortitis would be, in this kind, the absence of signs of mitral disease.

The physical signs of aortitis, which I have found convenient to discuss under the syphilitic kind, are to be discerned, with differences only of degree, in the other kinds. That aortitis should be sought and detected in early, acute, and often transitory modes, I am anxious again to impress upon the reader. Surely it is a serious matter to allow an aortitis in rheumatic fever, influenza, or other infection to escape our notice.

But there is one more physical sign, one sometimes of cardinal importance, the chafing sound of the dry, basic pericarditis, occasionally associated with aortitis. In all such cases therefore the sounds of pericardial friction must be vigilantly sought for. Thus we might detect an aortitis in rheumatic fever. Pericarditis about the aorta may arise in two modes: first, from the outside, invading the circumference of the aorta embraced by it; secondly, from within, whence aortitis may penetrate to the investing pericardium. If the former instances are mostly rheumatic, yet they may come of other infections; of Bright's disease and some other maladies. Now, whether the pericarditis be of inner or outer origin, it is, as I shall point out (p. 454), so likely to arouse angina pectoris that a nice appreciation of these conditions may be of cardinal importance. In some of these cases, as Pawinski [1] has demonstrated, in a remarkable essay from which I shall quote again in the Section on Angina, we shall see that angina pectoris is independent of coronary disease.

In a case of pericarditis sicca, described by Auscher,[2] three attacks of classical angina pectoris occurred, one of which caused

[1] Pawinski, *Deutsche Arch. f. klin. Med.*, Sept. 1897.
[2] Auscher, *Bull. Soc. Anat. Paris*, fév. 1896.

syncope. The patient recovered, but nine months later died of rapid phthisis. At the necropsy the effects of pericardial periaortitis were discovered; but to naked eye and microscope, the inner aorta, heart, and coronary arteries were alike "*irréprochable.*"

Enlarged veins on the chest are scarcely to be expected, apart from aneurysm. I need not say that the state of the pupils, retina, knee-jerks, and other ordinary tests, will not be forgotten. Thus in syphilis, as physical signs, we may have the tympanic second sound, the characteristic dull patch, the rise of the arch in the jugular notch, perhaps a rise upwards of the right subclavian: what else have we? for all but the first are late indications; long before it compassed these differences the evil was on foot.

Well, we have one more physical method, that of *radiography*, which may, probably will, give us more timely data. A good deal of valuable work has been done on this subject, and not without success. Vaquez and Bordet[1] have recently gone carefully over this means of diagnosis, and conclude that by the rays, using both screen and plate pictures, upright and recumbent, this latent mischief can be caught pretty early, and before the valve is injured. Vaquez's paper contains many careful diagrammatic outlines of such aortas, as seen under the rays, with tables of measurements. The points of diagnosis are that the shadow of the vessel becomes both wider and darker; points on which Lippmann agrees. In the Hamburg Hospital, Lippmann (*loc. cit.*) got positive results in 70 per cent of 180 cases. It is important to take pictures by orthodiagraphy, both sagittal and oblique, at angles of about 20-35 degrees. Vaquez believes that by parallel rays (3 yards off) "an exact and impersonal document can be obtained; the conditions being constant." But surely screen views in various positions should also be taken. In Vienna, Eisler and Kreuzfuchs[2] have come to similar conclusions; they divide their cases into the

[1] Vaquez et Bordet, *Paris méd.*, juillet 1911; and a book published 1913. See also Lippmann, "Röntgen-diag. d. Aortitis luetica," Ärzte Verein, Hamburg, *Protok. Deutsch med. Wochenschr.*, Nov. 21, 1912.

[2] Eisler, F., u. Kreuzfuchs, S., "Die R. Diagnose d. Aortensyphilis," *Deutsche med. Wochenschr.*, Oct. 30, 1913. See also Grödel, F., *R. Diagnostik d. Herz- u. Gefässerkhn.*, 1913.

Ascending-type, the Arcus-type, and the Descending-type; of these the Ascending is by far the most frequent. Mediastinal tumours, although they may oscillate with the heart's pulsation, may often, but not always, be distinguished by the outlines, if in the memory of the expert those of the aorta be clearly fixed beforehand. Stadler thus demonstrated a case (congenital) in a child aged 13. Aneurysm again presents its own distinction of outline; yet, as Ewald says, exclusion of aneurysm must often be difficult, and in case of small sacs near the heart impossible. Vaquez and Bordet give one obscure case in which syphilitic aortitis was clearly diagnosed by the rays, when otherwise physical diagnosis was at fault (p. 430). The dilatation and thickening were mostly below near the heart, as usually at the beginning they would be, and the summit of the arch was not pushed up; but by the rays increase of volume, alteration of curve, and darker shadow were apparent. Before the vessel is enlarged the rays can give no sign, unless possibly a reduction of the pulsations and a deepening of the shadow, indefinite signals. It is said that under specific treatment the shadow may be seen to lighten and the pulsations to return.

Such, and as yet so imperfect, are our physical signs of aortitis, a malady in its syphilitic form as prevalent as it is insidious, as stealthy as it is destructive. In cases where there are no data, in the present or in the past, to suggest syphilis, it is hard to see how we can be forewarned; we shall not even be consulted until some stenocardial sensation disposes the patient to seek advice. Then our course is clear; physical signs or none, we shall use the Wa. test promptly, and, if positive, press forward specific treatment with all speed and vigilance. Even with a negative Wa.R.,[1] if the patient's age and history be not inconsistent with syphilis, and if there be any percussion or ray sign, and no counter-evidence of any other infection, such as acute rheumatism or influenza, we must press forward the specific therapeutic measures, anxiously watching results. It is better in treatment, as we are wont to say of certain obscure

[1] In subjects of aortic syphilis, verified as such on the post-mortem table, as observers have of late become more and more expert, the Wa.R. is now stated to be constantly positive; but I presume that occasionally negative reactions, due for instance to the use of specific remedies, may still occur.

and possibly syphilitic cerebral conditions, to err on the positive rather than on the negative side.

How far the remedial effects of salvarsan may tell in aortic syphilis, with or without aneurysm, is a question now under active discussion. Mechanical injuries once inflicted may be irreparable, but some sanguine observers report under the use of salvarsan a reduction of the skiagraphic shadow. Let us hope they are right. Vaquez and Landry,[1] who have discussed the methods of treatment of the disease, report beneficial results from salvarsan in the majority of their cases so treated. Others, such as Gigaard (who has been quoted in various journals), still rely on iodide of mercury. The impression seems to be general that for this lesion the iodides alone are of less value (p. 126). A close watch is to be kept for any sign of relapse, on which treatment should be promptly resumed.

Aneurysm of the aorta, large and pre-eminent a part of aortic disease as it is, lies outside my subject (p. 168). I will only remind the reader how rarely it seems to arise from atheroma (Gee, Oppolzer, Hollis, Thorburn). Atheroma is not so directly a disease of the media, and it trends more to the floor of the arch; aneurysm inclines to the convex side of the arch. However, as a secondary effect, atheroma is found largely round the edges and bordering tracts of aneurysms, where it is to be distinguished from any syphilitic aortitis. Those aneurysms, too small for clinical observation unless possibly by the X-rays, which are apt to form about the root of the vessel, are often due to aortitis. In this area recesses are commonly found after death, especially in or near the sinuses of Valsalva; some of them are too shallow to contain clot, others retain clots of nominal substance; others again are narrow-mouthed pouches, aneurysmal in the saccular sense, and may rupture into the pericardium, or compress the base of the heart. These aneurysms occasionally give rise to signs of aortic disease, or to symptoms of angina pectoris (p. 431). Moreover, like gumma and other lesions of this part, they are apt, by interference with the conducting tracts, to retard the pulse rate.

In many cases we find a general arteriosclerosis with less eminent aortic disease, and may have moreover to discriminate

[1] Vaquez et Landry, *Arch. des mal. du cœur*, sept. 1912.

between the anginal symptoms of aortic disease itself, secondary cardiac discomfitures, and tabetic crises.

Atheroma of the Aorta.—The *morbid anatomy* of this disease of the aorta is for the most part included in its chapter in the essay on Arteriosclerosis (vol. i. p. 508). All the earlier records (Morgagni,[1] J. P. Frank,[2] Broussais) are or may be vitiated by the interpretation of intra-aortic post-mortem staining as an inflammation. Virchow [3] showed that the descriptions of "false membranes" on the inner face of the vessel were fallacious. Laennec and Guéneau de Mussy supposed aortitis, first perhaps described definitely by Cornil and Ranvier in 1869,[4] to be, like atheroma, a degenerative process; but Cornil and Ranvier described the gelatiniform patches with proliferative activity of the surrounding cells. If, anatomically speaking, the gelatiniform patch is a character of acute aortitis, we must not forget that this feature may be seen in active stages of chronic atheroma. In infective aortitis these fresh patches may be found full of microbes, especially of cocci.

We have seen now that aortitis is no uncommon event, and that even periaortitis is not very rare, and arises under many and various conditions. Now all diseases of the aorta, if they pass into chronic states, tend to atheroma; or at any rate to a blend of inflammatory with atheromatous processes. When ultimately syphilitic arteritis mingles with atheroma, the two diseases co-operate to produce the expanded, bossy, scarred, wrinkled and puckered vessel, together with the fatty and calcareous decay of the supervenient atheroma. I have referred to a good picture of the process in Letulle, vol. i. p. 156. The age of the patient is of course a factor in this transformation.

Rupture of the aorta is most commonly indirect, the rupture being of an aneurysm, large or small. Acute perforative disease, such as in the case of Oliver and Woodhead, is very rare; and, save under some striking circumstances, must be beyond diagnosis. But rupture of an aorta, apparently sound, or in a

[1] Morgagni's Twenty-sixth Letter.
[2] Frank, J. P., *De curandis hominum morbis*, Mannheim, 1792.
[3] Virchow, *Pathol. Anat.* vol. i., "Entzündung d. Aorta."
[4] See also *Histol. Pathol.*, 1881.

comparatively young person, as described by Morgagni more than once,[1] if an infrequent is not now a surprising event. Since Broca published his leading paper (*Bull. Soc. Anat.*, 1850) many cases have been submitted to the proof of dissection. In some the vessel, though not diseased, has seemed thinner than normal, as if imperfect in development. Pilliet,[2] in the same journal, added twelve cases to the list. Dr. Teacher showed me two such specimens in Glasgow. In both the rupture was about ¾ inch above the valve, and no proof of disease was found. In Dohrn's[3] case the vessel was found healthy. The rupture was clean across the arch on its downward turn at the point of attachment to the spine. It happened in a boy, æt. 18, who fell down 17 yards. There was no outward wound of the chest. The tear was as if due to a forward and downward jerk. But some cases are syphilitic; as for instance that of a woman (positive Wa.R.) in whom a circular rupture was discovered 2 cm. above the valve.[4] Here I may recall Letulle's opinion on syphilitic atrophy. Fränkel[5] gives four cases: (1) Male, æt. 68, cardio-renal disease and high pressures, and an atheromatous aorta (see p. 303); (2) Male, labourer, æt. 27; (3) Male, æt. 33, tuberculous; and (4) Boy, æt. 1½ years, ill with mild bronchitis. The accident was suddenly mortal in all. The boy had stenosis of the isthmus. In the essay on Angina (p. 433), I shall discuss these supra-sigmoid cases. Sir W. Osler[6] mentions one, in which from a first rupture the patient recovered, to die a year later of a second. A drawing of the aorta was shown. The first attack was diagnosed as angina pectoris, and rightly so. An effort or wrench has often determined the moment of the rift, which may not penetrate all coats but, checked by the outermost, form a dissecting aneurysm, as in a case by Pallasse and Roubier.[7] In the same issue of that journal cases were cited by Cairmont and others);[8] in one

[1] *E.g.* Ep. LIII. 35.
[2] Pilliet, *Bull. Soc. Anat.*, 1889.
[3] Dohrn, quoted *Zentralbl. f. Herz- u. Gef.-Kranktn.*, Juli 1914, from *Zeitschr. f. Med.-beamte*, No. 7, April 1914.
[4] Quoted *Zentralbl. f. Herz- u. Gef.-Kranktn.*, Juli 1912.
[5] Fränkel, *Berl. klin. Wochenschr.* Bd. l. p. 795, 1913.
[6] Osler, Sir W., *Lancet*, March 26, 1910.
[7] Pallasse and Roubier, *Lyons méd.*, 1912 (quoted *Arch. des mal. du cœur*, p. 600, sept. 1913).
[8] Same issue of *Lyons méd.*

of them the hæmorrhage forced its way to the origin of the left subclavian and down the abdominal aorta. The seizure began with intense retrosternal pain passing down the left arm. The heart was normal. Romeick[1] says death is often sudden, and may be painless, though in some cases angina pectoris appears with great severity, and generally with extreme angor ("hochgradigem Angstgefühl"). The angina he tries of course to hook on to some supposed anterior coronary sclerosis. In three-quarters of the cases the rupture opened into the pericardium. From the aorta the issue of blood is usually more rapid than in rupture of the heart, and survival so much the briefer. In rupture on the plane of the arch, as distinguished from rupture of a pouch, it is remarkable that the rent, though usually transverse, may run lengthwise of the vessel; when the immediate cause would seem to be rather a distension of the vessel than the tearing away from cardiac attachments pictured by some authors. Dr. Petch of York published a case (*Lancet*, 1899), of a man, æt. 56, who died suddenly, in which the aorta had parted from the base of the heart, and the distal end was retracted into the arch of the vessel, and invaginated after the manner of an intussusception. Other instances of this form are on record.

Of the *treatment of aortitis*, which indeed is almost altogether that of the general disease of which it is a part, there is little to say which will not be found in the sections on treatment of Arteriosclerosis and of Angina Pectoris; but, on account of the possible enfeeblement of the heart (p. 178), I suppose that salvarsan can be used only in persons otherwise favourably disposed, and then with every precaution. This I am told is Ehrlich's opinion.

Developmental deformities of the aorta, such as stenosis of the vessel at its duct, or at the seat of the ductus botalli,[2]

[1] Romeick, working under Marchand, *Spontane Ruptur d. Herzens*, Inaug. Diss. 1907.

[2] Botallo is to be held in remembrance as a great surgeon; but, so far as modern medicine goes at any rate, we owe our knowledge of this duct to the Bolognese anatomist, Aranzio. In ancient times it was known to Galen; as was the foramen ovale, etc. I may remark that the dilatation of the vessel

scarcely fall within my subject, and on them I have nothing fresh to say. In respect of the hypoplasia studied by Virchow, and the peculiar nephritis which the great pathologist found to be not infrequently associated therewith, I may refer to an able paper by Ettore Chiarruttini in the eighth number of the *Clin. med. ital.* of 1900, "Le Anomalie di calibro dell' aorta in rapporto alle nefriti e all' ipertrofia cardiaca."

Josef Burke [1] of Vienna published a valuable discussion of the whole subject of aortic hypoplasia; a discussion based upon one hundred recorded cases. He laid stress upon this defect as but an eminent part of a general vascular hypoplasia; in most cases at any rate it is an arrest of growth, and associated with other imperfections of embryonic development; occasionally it is betrayed by correlative defects. Burke is of opinion however that this explanation does not cover all cases; there is a group in which the hypoplasia is rather to be attributed to disease; sometimes an infection, sometimes a general malnutrition. In these cases, in young persons, he found atheroma of the aorta, and even arteriosclerosis, to be no infrequent feature, and assumed that in them a phase of high arterial pressure had gone before. This is not probable. Burke discussed the sex incidence of arterial hypoplasia, and concluded that in woman ensue anæmias, in man cardiac diseases. He entered fully into the kind and mode of associated and secondary changes of the heart. Among his own cases he published one of the nephritis already mentioned; in this case, as in others of the kind, the endocardium and serous membranes were inflamed.

Of abdominal aortitis I shall speak in the Section on Angina Pectoris. It may give rise to violent cramps and pains in the abdomen, and so often simulates other lesions, such as mesenteric thrombosis, tabes, colics, and so forth, that a differential diagnosis may be very difficult. To judge from the lists of cases which now and then see the light it would seem that the necessary discrimination is not always used. Any of the disturbances which may stretch or irritate the sensitive mesenteric nerve

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beyond the stenosis should be no matter of surprise, when we remember the need of compensation for the "vena contracta," and of reduction of velocity increased at the point of stricture.

[1] Burke, J., *Deutsche Arch. f. klin. Med.*, 1902.

end-organs would, so far as the pain goes, simulate abdominal aortitis. French observers speak of "hypertension" in the dorsalis pedis as one of the signs; but, as I have said elsewhere, in the presence of normal general pressures this is very improbable. A contracted vessel may give indications of resistance (Russell) wrongly ascribed to the blood pressure (Vol. I. p. 77). To palpation the abdominal aorta may appear much dilated, and tender to touch.

One or two cases of a congenital dilatation of the aorta are on record. Little is known of the condition, if indeed it prove to have any definite and independent features.

Appendix I. (p. 156).—I have seen and examined Jeeves five years later with two colleagues. She considered herself well, goes for walks, visits her friends, and so forth, but was avoiding laborious work. She had a double aortic murmur, and enlarged left ventricle. There was still a large area of dulness at the upper third of the sternum, and to the right of it, and a dilated aorta was palpable in the episternal notch.

Appendix II. (p. 184).—While finally revising these pages a respectable married woman, aged 64, came under my care in the Addenbrooke's Hospital with aortic murmurs, direct and regurgitant, and hypertrophied left ventricle. As in my opinion "senile atheroma," the diagnosis suggested, rarely issues in regurgitation, I searched for other evidence and found upon the right leg two or three such scars, one especially clean cut, about 6 mm. diameter, quite typical. Owing to military pressure our clinical laboratory was shorthanded, but Dr. Mott was so good as to apply the Wasser-Mann test for me, with a "strongly positive" result.

SECTION II.—ANGINA PECTORIS[1]

CHAPTER I

INTRODUCTORY

In this secret and fell disease there is a fascination to which no physician is a stranger, a fascination in its dramatic events and in the riddle to be read. By angina pectoris the humble out-patient is for the nonce lifted up into the sphere of a Hunter or an Arnold; over him we endeavour to bring the old discordant and mutually destructive arguments into some consistency, ringing again the old changes on the old bells. I too am content to compose another tune on the old chime: still, I have the excuse, at least to myself, of an independent endeavour, if not, as I diffidently hope, to solve the old problems, yet at least to elucidate them by compelling our nomenclature, our technical terms, and our current phrases to declare themselves each for what it is worth; and, no longer drifting hither and thither about the nosological field, to compel them to take up each its own rank, and no other than its own rank, in the argument. In this Section therefore some controversy is unavoidable; and as I must withstand adversaries better equipped than myself, I fear lest what Jurine said of Parry may be said of me, "Il nous semble que M. le Docteur a moins manqué d'indulgence

[1] The whole argument of this section was in substance contained in a paper read by me before the East Anglian Branch of the British Medical Association at Yarmouth on June 2, 1894; and, two years later, was incorporated in my Lane Lectures at San Francisco. It occupied a part also of my Cavendish Lecture in 1903; and almost in its present shape was read at the Fitzroy Square Offices of the Mount Vernon Hospital on January 9, 1908, or at any rate was so written for this occasion, though in the reading much of the subsidiary proof was necessarily omitted. It was delivered again, with some further illustrative matter, before the Section of Medicine of the Association at the Belfast Meeting in 1909. I republish it now in full, after revision.

pour lui-même que pour ses collègues." I would not forget Harvey's saying, "Concordia res parvae crescunt, discordia magnae dilabuntur," but I want the concord on my own side.

For concerning angina pectoris, more perhaps than any other disease, it is of importance to be quite clear at the outset what we are to talk about. It seems as if the perplexity of thought, so conspicuous in its interpretation, were infecting the clinical idea itself, dissolving it before our eyes. As in their case-records authors become more confused the more they grow bold to assure us, frankly or implicitly, and not without a certain complacence, that there is no such disease; moreover they not only disintegrate angina pectoris, or what was angina pectoris, but dress in the rags of it other and alien conceptions, after a fashion which cannot but destroy the stability of all clinical ideas whatsoever. In an editorial article in a recent number of a leading English medical journal, I read—not, as might be supposed, in irony—this concerning angina pectoris: "Its generally admitted symptoms have no fixed pathological significance; they may be associated with grave organic disease of the heart and coronary arteries, or with nothing more serious than a heavy meal, a disordered stomach, or excessive smoking"—or, he might have added, a squall in the nervous system. In other words, trivial and mortal disease are much the same thing, and technical terms may be blown about like straws in the wind. Because the pathologist —who year by year has been drifting farther and farther from clinical medicine—because the pathologist has been unable to demonstrate its anatomy, therefore the physician is to dismiss angina pectoris as a dissolving view. But not in this malady only have we to repair clinical ideas delivered from our forefathers but damaged in modern pathology.

Thus of such modern authors more than one, and these not the least distinguished, breezily assure us that angina pectoris is "not a disease but a symptom," a riddle over which I confess I have not perplexed myself overmuch. For how can one dwell on a statement so odd and so narrow when, within the very same pages, we may read under this same title descriptions of a series, of a many symptoms! "Symptomatic" angina may be; it is no doubt the symptomatic or clinical side of some internal lesion, as yet undetermined; but to speak of a series as a single term

is to confuse confusion. Other authors, fortified by the great authority of Latham, though equally determined to deny to angina pectoris the title of a disease, do admit that this name signifies a "symptom-group," a phrase with which, hackneyed as it is, there is less reason to quarrel; under it we may conceive some definite image. Yet for several reasons the term is inadequate: the word "group" fails to suggest a fairly uniform series or dynamic procession of symptoms. When is a disease not a disease, but a symptom-group? One writer has told us; he says "a symptom-group differs from a disease in its various causation and various pathological bases," which surely is to propound that a similar series of events can have dissimilar causes! If a single symptom, a cough for example, a pain in the side, or a hemiplegia—for a symptom does not become a disease because it is a big one—may be roughly attributable to various causes, it is unthinkable that an array and procession of events can issue from causes in the main different. Even within this narrow limit the acute and expert physician may perceive that coughs, pains, and hemiplegias of several origins are not indeed in character quite identical. Vomiting, for instance, usually betrays by its behaviour the kind of causes to which in the particular case it is due; and as we turn from symptoms to symptom-groups, the correlation of causes and consequences becomes more and more inevitable. The difference between a "symptom-group," or "syndrome," and a disease, if one may venture to interpret other people's notions, is that a "symptom-group" is a certain shorter chain of symptoms common to more than one completer series—a path of inosculation. To disregard the perspective of the phenomena is to lose the clinical idea; thus the picture of the malady as a whole, as in the case of angina, convincingly delivered to us by the elder physicians, is dissolved. Is not then all this hair-splitting between symptom-group and disease factitious? Is it not *chercher raison où il n'y en a pas*? Bluntly and tartly to deny to angina pectoris the name of disease, a word which, by the immemorial associations of thought, does signify to us a clinical picture, a morbid process seen in perspective, makes us wonder what is to be the content, colour, or compass of the word disease, which in this case is declared to be fallacious? What are the

notions now continually prompting certain able physicians to forbid us to call angina pectoris a disease ?

To answer for them is not easy ; we may best approach it by asking the larger question—What in any case do we properly mean by the term Disease ? It will be strange if in the twentieth century we find ourselves obliged to begin by protesting against a recrudescence of the scholastic ontology which supposed disease, and for that matter many another series of phenomena, to be each one a thing in itself. It is nowadays disconcerting, to say the least of it, to hear physicians of learning and experience gravely enquiring of each other if this or that malady is a "morbid entity," and implying that the title " disease " is to be reserved for " morbid entities " : no entity, no disease ! Thus to regard Disease is to regard it as a thing, or an immanent principle having an independent existence of its own, and to forget that it is merely an abstract conception of a multitude of similar experiences in the mind of a skilled observer. The judicious Latham was not very intelligible when he spoke of angina as not " a disease *sui generis*, but a series of symptoms " : for, clinically speaking, what is any disease but a series of symptoms recurring with fair uniformity ? Let us understand at once that angina pectoris, like typhoid fever or pneumonia, pretends to no " entity " ; that the name signifies a general idea of a process which takes place in certain sick men, never repeating itself identically—no two cases ever were identical,—but recurring with a measure of uniformity sufficient to make it possible and useful to construct for ourselves an abstract idea of it. Objectively, we may compare it to the photographic method of the superposition of many images of somewhat similar men, by which a " compound photograph " is constructed ; a picture not of any thing in itself, but an abstract notion of a certain group of approximate variations. Thus a certain disease, angina let us say, is a compound impression of the anginous phenomena severally observed in Johnson, Thompson, Wilkinson and others, and by the minds of Heberden and of his successors fused into an ideal picture, and by them communicated to us. The individual patient is indeed a thing, an entity if we please, but he cannot be named Angina pectoris ; this name signifies a general idea or concept, of which he is but a particular and a partial instance.

Now unless by comparison and revision we bring such individual notions into a common form, we can never agree, even to differ; for these mental concepts, or Diseases—Measles, for instance, Epilepsy, Appendicitis, and so forth—are no smooth and rounded images of fixed evolution; nor are they even definitely logical categories, within which all the individual instances of J. and T. and W., etc., can be accurately fitted; all "diseases," as is the case with all categories in biology, overflow each other, interpenetrate each other, and borrow each other's characters in ways which are those of nature rather than of logic. I have often said, figuratively, that, by the stepping-stones of borderland cases, one may walk from any one disease round the whole continent of morbid principalities and return dryshod to the starting-point. The title of Disease, then, is not to be withheld from a process, which on the whole is fairly consistent, merely because a few equivocal instances may refuse for a while to come in. Were this objection allowed, no classification in nosology could be maintained; for in all its classes aberrant instances occur abundantly. Notwithstanding, if in a large majority of cases we note that a morbid process recurs with fair consistency, if we are to reason of it, and if we are to compare it with other series, we must form a concept of it, and our concept must have a name. When Latham says epigrammatically, "We are sure that [angina pectoris] is an assemblage of symptoms; we are not sure what it is as a disease," the point, if there be a point, is soon blunted; for the same may be said of epilepsy, or gout, or asthma, yet who but a pedant would deny to any of these the name of a disease?

By a "disease," then, we mean no "entity," no "real" existence; we mean but our concept of a certain recurrent series of morbid events in many living bodies; and our distinction of any morbid process as a "disease," and by a name or label, is at most but a matter of convenience. If in respect of a certain series of events, positive and negative, by the general consent of competent persons it is agreed that the recurrence is sufficiently uniform to make a concept, and to justify a label, we label it; but, this done, such labels must not be shuffled. If at times, when we are diverted by the infinite mutability and continuity of nature, and are dwelling rather on the transitions and inoscula-

tions of her processes than on her uniformities, if under such impressions we begin to think that the examples classed under the label of angina pectoris, or under any other such label, prove too irregular in manifestation, too capricious, too eccentric to be fused into a type, then the indisposition to trim such cases into a category, and to depict and to label a general concept of them, is put upon proper ground, the ground of inconvenience. In my opinion however, an opinion I believe based upon good historical doctrine, the process named by Heberden in 1772 "Angina pectoris," is, in comparison with other morbid series, even with the most definite of them, a remarkably consistent one; and its characters even peculiarly distinct and impressive. An able American physician, who hesitated to allow to angina the place of a disease, admitted nevertheless that, "clinically speaking, angina is an important and strongly individualised disorder, with striking characteristics, and a clinical history peculiarly its own!" Why, what more than this can we as clinical physicians want for the recognition of any disease, or to justify any name in nosology?

I submit therefore that our examples of angina pectoris have common and characteristic features such as to enable us to form a general concept of it, and to attach to the idea a label or name. And, for the series it has represented from Heberden's day to our own, the name of Angina pectoris is a convenient one, for happily it commits us to no doctrine.

Although Heberden rightly enjoys the place of godfather of the disease, as he was the first to publish a formal description of the series and to name it, yet more than once the disease had been incidentally adumbrated before;[1] as by Morgagni,[2] by

[1] *Med. Trans.* vols. ii. and iii. The history of angina is not dealt with in these pages; it is compendiously given by Gibson in a lecture published in the *Edinburgh Medical Journal* of July 1902; but it is fair to say that although Heberden named the disease and gave by far the better description of it, yet the first precise discernment and description of it was in a letter by Rougnon to Lorry, published at Besançon by J. F. Charmet in 1768. Pawinski (*Zeitschr. f. klin. Med.* lxx. 3-4) says the disease was described by Baillon in the sixteenth century. Still, the conception of angina pectoris may be said to belong to England. Heberden's description was published five months later, it is true, but it was so acute and comprehensive that, while later authors have tried their best to confuse it, they have found little to add to it.

[2] The most apt instance cited by Morgagni is in the 26th chapter (xxvi. 31); but he makes interesting allusions to the disease in several passages.

Willis [1] under the general head of Asthma convulsivum ; by E. A. Müller [2] as Asthma occultum siccum—asthma was confounded with angina by our forefathers as scarlet fever and measles, and as typhoid and typhus, were confounded, and as cardiac dyspnea and asthma are confused to-day—and by Hoffmann (in his *Medicina Consultatoria*, early eighteenth century) as Præcordial asthma. Hoffmann's [3] example was a definite case of the disease. Senac, under the head of heart diseases, speaks of attacks of pain, " Cujus paroxysmi summe procellosi majorem incutiebunt terrorem " ; moreover of a certain patient " pectoris anxietate molestissima laborabat." Morgagni first described the brachial numbness and the aortic lesion. By writers such as these the series was gradually defined and named, and so the matter stood until, in our own generation, alien processes were stuffed into this clinical conception, whereby the standard idea has been blurred and the attention of the student disconcerted. This muddling of standard clinical concepts is a new temper, and to be seen in other clinical fields. Thus into Epilepsy certain loose thinkers are thrusting series of other kinds, syphilitic or uræmic for instance, merely because, although of alien nature, they are attended by convulsion ; into Asthma, as I have said, any paroxysmal dyspnea : and so again under the labels chorea, chlorosis, etc., diseases alien in nature are intruded. To rest content with our patterns would be a contrary error ; we must always be revising them, winnowing the casual from the primary and cardinal phenomena, and detecting deeper and deeper affinities. Moreover some names and types of recent mintage, such as neurasthenia or rheumatoid arthritis, which have been long under criticism, may on good grounds be called again and again into question, and certain particulars redistributed. But when one of the most distinguished of living physicians writes as follows, " One may still

[1] On p. 541 col. *a* of the edn. of Willis in my possession (Venet., 1708), the case is " Senex perhonorificus, multis magnisque titulis insignitus, iisdem omnibus major," who complained of a paroxysmal pain at midsternum—attacks often at night—without dyspnea (" sine quavis tamen dyspnœa ") " or other sign of asthma." For these reasons the case was regarded as exceptional ; asthma Willis attributed to spasm of the muscular tissue of the bronchi, and of it dyspnea is an essential or primary feature.

[2] *Op.*, Geneva, 1736, p. 439.

[3] Hoffmann, *Op. Omnia*, Geneva, 1753.

read of angina pectoris as a separate and fatal affection of the heart, instead of its being regarded as a painful incidence of varied significance that may occur in the course of a wide range of functional and organic diseases of the cardio-vascular system," do we not perceive that there is a danger in looseness of speculation as well as in tightness of definition ?

In a roving quest the eye is too readily caught by prominent phenomena or analogies which may have no deep significance, while the inward affinities of things are overlooked. Thus if to call a whale a fish be consistent with good poetry, it is certainly inconsistent with good science. So, speaking generally, it has been of late with angina pectoris : instead of penetrating to its deeper affinities, dwelling upon these, comparing them with the deeper affinities of similar processes, and classifying them severally by the profounder likenesses, too often the eye of the modern physician is so caught by shallow analogies, by taking and superficial semblances, that incongruous groups or heaps are thrown together with a shovel. For instance, in a recent interesting article, the writer, studying the anæmia of tuberculosis and demonstrating truly its deeper qualities, contrasts them with the deeper qualities of chlorosis, so as to illustrate their profound dissimilarity; and yet in conclusion he allows himself to name this anæmia " tuberculous chlorosis " ; that is, he continues on superficial likeness to classify together processes which it was the purpose, or at any rate the purport, of his article to contrast and divide on lines of intimate difference. If such is to be our way of naming, all nomenclature, all classifications indeed, must come to confusion ; and the laborious differentiating work of clinical medicine, from the time of Hippocrates to this day, will be brought to nought.

Still, dismissing all these distractions of meaning, the reluctance of modern teachers to concede to angina pectoris the title of a Disease is not quite accounted for ; there must be some grounds, good or indifferent, for the demur. If indeed it were true that angina pectoris is " only a symptom," this denial to an unattached phenomenon of full recognition as a disease would be justifiable. One symptom—a toothache, for instance—may enter into many a clinical series, and be a criterion or a constant of none. When I am told that hemiplegia is a " symptom-

group," and yet not a disease, I regard it as neither: I have said that a symptom does not become a "symptom-group" because it is a big one; hemiplegia is but a symptomatic event in the course of several diseases, such as granular kidney, hyperpiesia, epilepsy, poliomyelitis, and so on. As I have said, it may be regarded as a few terms common to several diseases; two or more having, for a short distance, what railwaymen call "running powers" over the same line. But if, as I shall argue, and as we shall all have to admit, angina pectoris be not a symptom but a complete morbid series—that is to say, from beginning to end a fairly uniform association and succession of many symptoms positive and negative, and if by the word Disease we mean such a series,—and what more by it can the physician mean?—then angina pectoris occupies its place in our minds as a characteristic clinical concept, and our convenience dictates for it a particular label. Yet notwithstanding, as this reluctance so to accept it continues, an excuse may be found in the shifting explanations of the pathology of the anginous process. It is true we are not yet agreed upon the pathology of this disease, whether static or dynamic; but are we not still more ignorant of the pathology of Chorea, of Epilepsy, of Pneumonia, and of many another series to which, without a breath of dissent, we attribute uniformity enough to justify definite titles as Diseases? And is not angina pectoris as consistent a series as any of these? The books, only too unanimous in their consistency, copying one from another both errors and truths, endow it with even more uniformity than by nature it has. For instance the text-books, almost without exception, state that the pain in angina pectoris is "præcordial"; whereas in the vast majority of cases it is in the upper sternal area: præcordial pain in angina is rare, and generally incidental.

Does it not seem then incredible that a series of events so grave, so complex, so complete, and so fairly uniform should consist now in one chain of causes, now in another and widely different chain? Surely we must concede that as a clinical series approaches uniformity its causes must also be approximately uniform. Caution in accepting any alleged chain of causes as that on which angina pectoris depends is a scientific suspension of judgment; but eagerly to contend that so signal an

evolution of peculiar and menacing features may be caused now by this set of antecedents, and now by that or the other widely different set, is surely not suspension of judgment, but a suspension of thought itself? Dr. Colbeck well expresses the opinion, which he kindly attributes to me, that if the title of angina pectoris be used to include a variety of symptom-groups which, although they may present a superficial clinical resemblance, yet in their etiological and pathological relations differ widely and fundamentally, all definite use of names comes to an end.[1] No, angina pectoris, as it has its peculiar features and march, must have its own independent and definite pathological basis and interpretation.

[1] Colbeck, "Angina pectoris," *Lancet*, March 21, 1903.

CHAPTER II

SIMULATIONS OF ANGINA PECTORIS

Mock Anginas.—Let us then, if we are to understand this disease, determine to repudiate what I have called classification with the shovel, and, discarding superficial resemblances belonging to other and widely different issues, return to classification by intimate affinity. Let us dismiss from co-ordinate classification all "pseudo-anginas" and other echoes, which from this point of view are will-o'-the-wisps.[1] I cannot undertake to describe all the phantoms, whether genuine nerve storms or fanciful, and even ephemeral phenomena, which have been piled upon, and so overlain, the conception of angina pectoris as to hide from us its austerer features. Elaborated tables are dressed up, as it were for students' examinations, of the marks of distinction between false anginas and the true; tables of which the valuable lesson is not their resemblances but their polar estrangements. And meanwhile, not invisible to the eye of the discerning physician, through all this crowd of competitive disorders—complaints "which," as Broadbent with playful exaggeration used to say, "never interfere with a pleasant engagement"—the pale horse of the mortal disease takes its inexorable way. The appearance in these dreadful lists of unsubstantial functional disorders seems fantastic. I do not say that none of the mock anginas is grievous, some of them if not very alarming are distressing enough and importunate enough; nor do I assert

[1] Dr. R. O. Moon and some other writers (*Clin. Journ.*, March 12, 1913) call the mock angina, which Dr. Moon fully recognises as false, "Angina minor," which unfortunately signifies a process of milder degree but of the same nature. But with much else that is useful in diagnosis he does not fail to repudiate the pseudo-anginas. I still call "stenocardial oppression" (p. 287) A. minor, because, though milder in degree, it is of the same nature.

that from them nothing is to be learned for the interpretation of some secondary phenomena of angina pectoris; but to confound these alien series with angina pectoris itself, and to give them this solemn name, even with a difference, seems to smack of the ingenuity of scholastic logic rather than of that discernment of premises and origins which is born of scientific method. For when we essay to compare the mock anginas with angina pectoris, as in the world of practice we see it and them, they are manifest as fakes. As a student of cardioarterial disease, towards whom there has been a drift of cases of this kind, I do not hesitate to say that a physician of experience should not feel more than a passing doubt concerning any one of these illegitimate claimants. And as a name Pseudo-angina will never do; no category can be founded upon negation. Sir William Gowers, whose insight into the deeper relations of disease is so highly esteemed among us, has swept out of our way the mock anginas, with the other more important of this kind of comparatively trivial maladies, by putting them into a family of their own, under the significant title of Vaso-vagal Disorders.[1] There let them stay, while in the meantime we may learn from them new lessons in pathological kinships.

Still it is of such primary importance to realise precisely what we are to discuss that I must be forgiven if I insist even a little longer upon this purging of the subject. If the reader will devote a few hours to a perusal of the older writers on angina pectoris, of the authors of the end of the eighteenth and beginning of the nineteenth centuries, with their truer sense of clinical form, as he returns to modern medical writings he will note with some bewilderment that he is leaving the solid ground of the natural history of disease for the quagmire of clinical sophistry. Heberden, Jenner, Fothergill, to name but a few of the more familiar authors of the eighteenth century, not only kept a watchful eye upon the patient, but kept also a due sense of the relative values of the facts, an appreciation of the facts that matter; whereas the more rolling and speculative eye of the modern professor is apt to wander. Accordingly all these observers take for granted the truth that in every case of angina pectoris there is some grave lesion, and that in the very few necropsies published as negative some such lesion had been over-

[1] Gowers, Sir W., *Lancet*, June 8, 1907.

looked. That angina pectoris is a definite organic disease of the utmost gravity our forefathers never forgot; accordingly, in this seriousness, they charted its symptoms and learnt its signals the more accurately, and were not easily carried out of their course by mirage. Our forefathers had to hug the shore; we who fetch a wider compass are tempted to sail out of our bearings. In making their diagnoses of angina they weighed not only this feature and that, but the whole series to which they belonged, and the issues to which they tended. And, in the next generation, Latham thus sagaciously mistrusted the notion of a mere functional, or "nervous," angina pectoris. He named the counterfeit angina—angina notha; but it is better both for doctor and patient to avoid for it the name angina altogether. These observers were not beguiled by such equivocal phenomena as palpitations, arrhythmias, pantings, nervous flusters and crises, and other spectres of the mist; and, guided by pilots so sagacious, the reader feels secure of their evidence and their judgment, so far as they go. The extraordinary acuteness of Morgagni was here again not at fault; after speaking of high arterial pressure as a plenitude in which the arteries could neither be more distended nor contract upon their content, he says there is a pressure of another kind:[1] "at another time we may perceive (a different kind of plenitude) the coats of the arteries contracted by the nerves, which is especially apt to occur in hysterical patients, the arteries being then reduced to mere filaments, so far, indeed, that in some regions the pulses are suppressed, and these parts turn cold while other parts remain warm." He adds that these phenomena may supervene in graver maladies also. It is with a different apprehension, with a sense of equivocation, that we follow the erratic course of some essayists on angina of our own day, who drift in "unaccountable diagonals between truth and falsehood" —between, in this instance, angina pectoris and the masquerades of it which, as Dr. James Mackenzie well says, are only "cloaks for our ignorance"; ignorance the less venial as those forefathers, who saw at least as much of gusty nerves as we do, had left

[1] Morgagni, *De sedibus*, lib. xxiv. c. 12, "alias ob arteriarum tunicas, ab nervis contractas, ut in hystericis praesertim contingit, cum arterias ad tenuis filamenti modum redactas percipimus, imo et pulsus certis in partibus supprimuntur, unde tunc illae, reliquis calentibus, figent."

their charts behind them for our guidance. It has been well said, on another matter, that "the continual use of a nomenclature in which essential distinctions are ignored closes the mind to the very existence of those distinctions."

Lartigue[1] is claimed by French writers as the first influential physician to fog the picture of angina by throwing these cross lights upon it; but Forbes had fogged it before him.[2] Forbes declared that in about 50 per cent of cases of angina there was no organic disease, and that in the rest it was associated with all manner of organic cardiac and aortic lesions. To Landois (1863) belongs, it is said, the doubtful credit of having dragged the vasomotor red herring, and invented the misleading title of Angina vasomotoria, or reflectoria. Walshe was tempted to follow; but it is to Nothnagel[3] chiefly that we owe the transcendent confusions of recent authors. No wonder the descriptions of angina vasomotoria are various and inconsistent; that for example, as Dr. Mackenzie says in his work on "The Pulse," the descriptions of Sir R. Powell and Nothnagel bear no mutual resemblance. How far Nothnagel had wandered, and wandering led others astray, I did not myself fully comprehend till I consulted the article on Angina pectoris—or on the ghost of it—in von Ziemssen's *Encyclopædia*. This article, guaranteed as it is by a distinguished name, can scarcely be acquitted nevertheless of perversity, and even of levity; for surely it is levity to confuse the squalls—*nervöse Angstzustände*—of unstable "neurotics," mostly women, with the assault of one of the fiercest and most searching afflictions which can fall upon steadfast and resolute men. It is scarcely credible that in this *Encyclopædia* under Diseases of the Circulation angina pectoris has found no place! After long search I discovered it at last elsewhere, under the fanciful headline of "Vasomotor and Trophic Neuroses," and placed between such strange bedfellows as Progressive Muscular Atrophy on the one side, and Graves' Disease on the other. Could professorial pedantry be more extravagant! unless, indeed, it be surpassed in this very article by a comparison of the pain of angina with

[1] Lartigue, *Mém. sur l'angine de la poitrine*, Paris, 1846.
[2] Forbes, *Cyclop. Pract. Med.*, 1832.
[3] Nothnagel, *Deutsche Arch. f. klin. Med.*, 1867.

that of colic, of "hysteralgia," or of heartburn ("cardialgia")! But the most egregious example of this eccentricity which I have noted is the description, by a well-known modern physician, of a case of "angina pectoris originating in the clitoris!" Such is the exorbitance to which by looseness of language we may fly; for surely to confound spasms, throbs, and vapours such as these with an affliction so awful as angina pectoris, is, I may reiterate, to lose sense of the relative value of facts, to ignore in the stream of function the facts that matter, and to take analogies and shallow semblances for natural affinities.[1] The vasomotor derangements which form the staple of the lapses which, after Sir William Gowers, we will call Vaso-vagal Disorders, are, it is true, no uncommon accompaniments of true angina, as they are of other paroxysmal irritations of afferent nerves, and vexations of corresponding spinal segments. One claim to consistency indeed that encyclopædic article can make in its scarcely veiled grudge against certain unconformable facts of the very disease which is its nominal subject; namely, of angina pectoris itself.

Against such fantasies naked sense is an unequal adversary, so it is a matter of regret rather than surprise that some contemporary, and not inconsiderable, English writers on this subject have been so influenced by them as in their turn to admit under the title of Angina pectoris a like medley of heterogeneous disorders. Even Gibson was bold enough to opine "That true angina pectoris results from reflex, vasomotor, neurasthenic and hysterical causes is now beyond the region of doubt!" But if we are to place "true angina pectoris . . . beyond the region of doubt," we must begin by stripping the "true" idea of false conceits. What would my lamented friend have said of me had I classified functional and organic hemiplegia together, or put hysterical convulsions with epilepsy!

Now *the vasomotor phenomena of angina* may be discriminated

[1] Pal's brilliant tract, seductive on a first perusal, is so largely hypothetical that it does not gain on further acquaintance, but loses rather. Many of the cases quoted are of dubious diagnosis; and we are often left in doubt whether rises of pressure were cause, effect, or correlation. Indeed one begins to doubt if it contains more than in a pedestrian fashion we knew before. Why for claudication is any spasm necessary? The condition is one of reduced potential; depleted stores, often due to damaged vessels.

as (*a*) the *Secondary or Concomitant*, and (*b*) the *Accessory*; of primary importance there is none (p. 283). The Secondary symptoms are the fringe of the case, and may not concern the patient very much, if at all. We all know the pale face and cold extremities of renal colic; nay, every man who, when he is hurt, flushes or turns pale is familiar with this vasomotor sequence of pain. By various repeated or severe irritations of sensory areas we may call forth secondary vasomotor disturbances, almost at will. The Accessory, those which consist in such intercurrent contingencies as chill or emotion, if they may determine the moment of an attack, no more constitute it than the detonator constitutes the torpedo; but of these epiphenomena I shall say more presently (see p. 340). Thus Dr. Morison agrees with me that in this way a rise of blood pressure may be the consequence of an attack of angina, as at other times it may be a proximate and determining cause of it. For instance, on another disease, an author, whose book lies on my table, writes as follows: "On rubbing the painful area the pulse became very small, the face ashen, and sweat arose in the hands and axillæ." We all know how commonly an aching in the areas of the 6th to the 8th dorsal nerve on the left side is associated with states of debility and vasomotor disturbance; and not in women only. So long ago as 1870, at the meeting of the British Medical Association at Newcastle, I commented upon cases such as this: "A young woman complains of tingling, numbness and weight in the left arm and leg. She fears paralysis. She has also pains radiating about the brachial plexus, including the intercosto-humeral branch. When the pain is severe, arise also heats and chills, flushings, strange visceral sensations. On enquiry we find she has lately been confined, is suckling a big boy, is menstruating, and at the same time has been troubled with diarrhœa and want of appetite. We note also characteristic tender points in the usual places, especially in the submammary intercostal area—a part from which such disturbances often take their rise, and where in this case the severer attacks begin; indeed she is never free from aching in this place." It was then more necessary than it is now to point out the distinctions between these cases and organic disease, whether cerebral or cardio-aortic; and are we to turn back to such confusions! A large proportion of the cases of "false

angina" under my notice have been associated with a more or less severe intercostal pain of this kind.

The paths then by which pains are propagated outwards may serve also for the inward penetration of external influences. Barié records such cases, and we may witness good examples of it every day. But—and this is an interesting reflection on my view of angina pectoris—while by radiations apparently from without inwards we often find that the heart is vexed, in anginal attacks, in which the aorta is usually the commencing term of the series, as a rule it is not. Thus in severe penetrating intercostal neuralgia, for instance, the heart is often much disturbed (see p. 227 *et seq.*); wherein lies one of the sources of a gratuitous confusion between such cases and angina:[1] gratuitous because cardiac disturbance is not a character of angina. Yet, if intercostal neuralgia be eminently capable of producing vaso-vagal storms, it is by no means alone in this capacity. Some years ago I saw in consultation a lady afflicted with a suppurative disease in the pelvis for which severe surgery had compassed but a partial relief; she was weak and emaciated, and suffered from attacks of violent but apyrexial pain in the right pelvis. One of these attacks I witnessed on my visit—it began with a general vaso-motor constriction, pain under the left breast, cardiac turbulence, panting, and much agitation. Vasodilatation and sweat succeeded the spasms. Many of the attacks consisted only in the intercostal pain, pain which in extreme attacks radiated into the brachial plexus. But the whole aspect of this side of her case was "neurotic." In many such cases there is no pain. Not long ago a lady had "one of her attacks" in my consulting room. As I was feeling her radial pulse, the artery, as Morgagni remarked, rather suddenly shrank to a thread, and then ceased to be perceptible. Tension rose high in the larger vessels; the breast and heart became oppressed and the respiration panting; but there was no pain. Such attacks often depend on urogenital, abdominal, or other visceral lesions or dislocations, or even on carious teeth.

[1] The signal of caution is never absent from the medical path. While writing these lines I recall Fräntzel's remarkable story of Jules Ferry, who died of angina after a wound of an intercostal nerve. The record as it stands, and so the diagnosis (*Herzkrankheiten*, p. 224), are however ambiguous.

In the following case again the vaso-vagal symptoms became detached from the pain :

Miss E., æt. 48—a pallid blinking spare woman. Dysmenorrhea till menopause three and a half years ago. Ardent temperament and full of active and useful works. Has had domestic trials and nursing cares for some time, but worse last two years ; pains in intercostal and brachial nerve areas. Violent pain, precisely in region of cardiac apex, radiates into left arm—"in the bone"—she indicates the whole thickness of the arm, especially the outside aspect. Also in shoulder blades and down the back ; never in the neck or face, but may reach the occiput. With the pain she becomes cold all over, and prostrated, and thus remains for 5-6 hours ; then the circulation "begins to move again," and warmth returns, followed by flushes and profuse sweating. But during the last few months the pain has wholly ceased, and only the vasomotor symptoms persist. The attacks are relieved by nitrites. Physical examination, etc., wholly negative, except some small patches of anæsthesia, and slight contractures. These used to be far worse, especially at the catamenial periods. I find no note of the visual fields.

In one of his cases of this kind von Basch found in the constrictive phase that the blood pressure rose to 170 and 200 ; a few minutes later it fell back to normal.[1] Pain or no pain, to call such functional vacillations as these by any name even distantly suggestive of angina pectoris is surely chimerical. Yet Sir Richard Powell,[2] whom, as one of the most eminent of living authorities on this subject, I cannot pass over, says : "In a large proportion of cases angina is an entirely functional disorder, the main feature of which is sudden increase of blood pressure, and a corresponding call on cardiac effort. . . . The vasomotory are the most intense forms of angina. . . . In many cases angina pectoris is dependent upon a disorderly action of the vasomotor nerves, and is associated with a sound heart. In many other cases we have a similar mechanism but cardiac disease in the background ("Secondary Cardiac Angina"). . . . In numerically a comparatively small, although very important, group the anginal failure is due primarily to Cardiac disease ; Syncopal angina or primary cardiac angina." I cannot but regard this

[1] This record may have depended in part upon resistance of the tight arterial coat. (See Vol. I. p. 72.)

[2] Powell, R. D., *Lancet*, June 29, 1901.

comparison as one of superficial, not deep, resemblances, of analogies not of homologies or of affinities.

Again, of the so-called "vasomotor" angina let me select another even more interesting example:

Mrs. X., patient of Dr. Mallins of Watton, æt. 52; seen November 1910; menopause over five years ago, without serious trouble. As on my visit she lies in bed, about midday, has the aspect of health both mental and bodily. A very intelligent, capable woman. No specific signs of hysteria. No globus. Plantar reflex, ovarian and cutaneous sensitiveness, etc., normal. Tendon jerks not excessive. In her past history the only relevant event is of rheumatic fever many years ago; without however any cardiac effects—as verified by Dr. Mallins, by Dr. Mitchell Bruce (whom she had consulted a year before), and now by myself. She leads a happy and placid life, and has had no worries. She tells me that, as she converses with me (about 1.30 P.M.), she feels quite well; but almost daily "about tea-time" attacks come on of a kind which, for a few years past, have increased upon her both in degree and frequency. As she lives at some distance from Dr. Mallins, the attacks have not been witnessed—not at any rate in their full course—by an expert eye; but she gives a clear description of them. Rather suddenly (as in Nothnagel's cases) she turns cold, very cold if out of bed, but in bed—where she has been staying for a few days as an experiment—with a less severe chill, a general coolness all over the surface of the body. About the same time she feels a "huskiness" in the throat (larynx ?) (no cough or expectoration at any stage), and the respiration becomes faster and faster until she is panting—in and out, not with a fixed chest—at a great rate; rapid shallow breaths. Meanwhile an oppressive weight gathers upon the upper chest almost to suffocation—she sweeps her hand over the whole breadth of the upper thorax above the breasts, and a pain arising "at the heart" strikes up the anterior left axillary line, but never enters the arm; of this she is sure, though it is prone to run up more or less into the neck and about the left shoulder (phrenic ?). Still the chief seat is "at the heart," and up the anterior axillary line. In answer to the question, "Where is your heart?" she points precisely to a spot in the seventh space about the size of a florin, well below the heart's apex and abutting upon the left costal arch. This spot, always tender to touch, during the attack is extremely painful, with a continuous acute pain. After an attack the touch of the stethoscope on the spot is for some hours intolerable. On investigation I found another definite tender spot also about the issue of the seventh thoracic nerve close to the spine; of this tender

spot she was aware, but it had not annoyed her so much as the anterior spot. During the chilly stage of the attack, besides the oppression and panting, she has an alarming sense of cessation of the heart's action. On being reminded that in health we ought not to be aware of the heart's action, she adds that the suffering is not negative, but " as if in stopping the heart gathered itself into a heavy ball." The temperature is normal throughout. Concerning any changes of complexion during the attacks there was no definite testimony.

After a while of this chill and oppression—perhaps twenty to thirty minutes, perhaps less, for in suffering time seems interminable—she flushes all over, the arteries begin to throb, and she tingles and burns with returning tides of hot blood bounding through the vessels; a stinging pulsating heat, uncomfortable enough but almost welcome in the rapid solution of the oppression at the chest, and of the panting. The attacks are followed by a sense of great prostration or exhaustion; but after a night's rest she feels quite well again.

At the time of my visit no dulness was to be detected about the arch of the aorta; the subclavian and other accessible arteries were soft, or impalpable; the blood pressure was rather below than above the normal; the artery was not then small or wiry. The heart was free from signs of disease, and betrayed no abnormality of stresses or of rhythm. The spleen was not enlarged.

This patient, in the exercise of self-control and common sense, had done her best to avoid apprehension of the attacks. She had often persuaded herself that the attack of the afternoon would not—should not—return; but in vain: determined, convinced, even preoccupied as she might have been against it, in spite of herself she was seized. She cannot think that auto-suggestion has any great part in the return; or that therapeutic suggestion would prevent it. The only possible toxic cause Dr. Mallins and I could suspect was a " rheumatic " tendency, of late years hanging about her in the form of " fibrositis "; especially in the fibrous structures of the knees and ankles. At the time of my visit, when she had been some days in bed, there was nothing to detect except a creaking left knee; though when about, and in damp weather, these parts were wont to swell. We had no hesitation in recognising her seizures as the " vaso-motor angina " of Nothnagel and others. The patient said that the initial disorder was the chill, the next " huskiness "; then rapidly ensued the oppression at the upper chest and the panting. This oppression, described as very severe, may have

been due to high tension of the aorta consequent upon extensive areas of vasoconstriction; and in a diffuse bulbar discharge the heart may also have been involved directly.

After emphatically reassuring the patient, we prescribed the administration of atropine in doses of $\frac{1}{100}$th of a grain twice a day. Ten days later Dr. Mallins wrote: "On the next morning she began the atropine; and on and from that day the attacks were reduced to quite miniature affairs, which, on the fifth day thereafter, disappeared altogether. This relief was at the cost of intense dryness of the mouth; so that I have reduced the dose to once in the day." To re-educate the vasomotor system, the practice of a simple hydrotherapeutical method was also recommended; but these measures—the season being winter—had scarcely been begun, and could not have had much effect in the first mitigation and final banishment of the seizures. But I may add that we had applied a small blister on the back, over the place of exit of the seventh dorsal nerve.

Now, taking these phenomena at face value, do such characters resemble angina pectoris even superficially? Have we any more reason to call these cases angina pectoris than to call the well-known throbbing of the abdominal aorta in women angina abdominis, or aneurysm? The so-called "heart pain," present in many, if not in all, of these cases, the sense of distension of the whole upper chest, often with submammary pain and intense local hyperæsthesia, are neither in seat nor kind like the pain of angina; the breathing, far from being held as in angina, is panting; the bodily state is not awe-stricken, but restless and agitated. Upon this contrast Huchard repeatedly and emphatically insisted. That angina pectoris, like any other painful disorder, may be accompanied by vasomotor disturbance, is true; so epilepsy may be, so may be migraine, so ague, and so on; while, on the other hand, angina pectoris may be, and indeed often is, quite free from these epiphenomena.

We have, all of us, a plenty of such cases in our notebooks, so I forbear to narrate many of them. Before Huchard's book was published, Peyer of Zürich[1] and other authors had demonstrated the differential features of these mock anginas.

[1] Peyer, *Wiener med. Presse*, Juni 16–26 and Aug. 2, 1892; very interesting papers.

In Peyer's practice many cases had been in males, often in anxious overworked students. In some cases he noticed the periodicity, as in Dr. Mallins' case; though more usually the attacks were irregular in recurrence, or were subcontinuous with exacerbations. Attacks sometimes came on in the night, *e.g.* 2 A.M. In young men seminal emissions and frequent micturitions were often present. Peyer also laid stress upon the diffused oppression of the upper front of the chest, upon the submammary seat of the lancinating pain, and upon the cardiac sympathy—palpitations, throbbings, and clutchings, as if the heart were too big; all, as he points out, being, like the dyspnea, features differential from angina pectoris. In such patients the whole history and bearings of the case are distinctive. And yet Gilles de la Tourette [1] proposes the pulse as a criterion! After saying that in the neurotic kind "rien ne manque au tableau," he offers as our guide that in the true angina the pulse is "small, miserable and even intermittent," but in the neurotic kind "ample, full, and regular." The difference between "ample" and "plein" is not obvious; and his diagnostic points of angina suggest that the learned author will command most attention in his own field. Curschmann [2] again, an accomplished physician whose death we are regretting, wrote likewise that in pseudo-angina all the symptoms of the genuine cases occur ("alle Symptome wie bei echter Angina Pectoris"!). To prove this he quoted rapidity of the pulse, palpitation, pain at the heart, and sense of oppression to the degree of losing one's self ("Vernichtungsgefühl"); none of them characters of angina, in which, by the way, the mind is generally only too clear. He added that in vaso-vagal attacks the pulse never disappears from the wrist. Twice at least in such cases I have felt the pulse slowly diminish till it became impalpable, and the patient "far off," or transiently unconscious (p. 227). I may observe that in many of them the crimping of the vessels seems, at any rate in large part, to be musculo-cutaneous. Conversely, in his argument against Huchard, Curschmann describes cases of "Vasomotor angina," of which three were fatal, and after death presented coronary arteriosclerosis, to prove not that, like the

[1] G. de la Tourette, *États neurasthéniques*, Paris, 1898, p. 23.
[2] Curschmann, *Lehrbuch d. Nervenkr.*, 1909, p. 832.

rest of us, he made mistakes in diagnosis, but that pseudo-angina and true angina are the same disease! Upon the symptoms of these fatal cases he was judiciously silent. Weintraud, although writing in Eulenburg's *Lehrbuch*,[1] speaks with far more clinical discernment, not being a "nerve-specialist." So far, in his opinion, are the vaso-vagal attacks from "presenting all the symptoms of the genuine angina" that he draws a clear contrast between the two maladies, and points out that "the spurious form is merely a nervous malady which has nothing whatever to do with heart disease." Gallavardin[2] first classifies the mock with the true form, and then labours to show how profoundly they differ! Neusser (*loc. cit.* on p. 303), though the very inventor, I believe, of the coronary cramp conjecture about true angina, if he does not discard these false anginas, says frankly, against his compatriots, that "The majority of these forms have only in few features any similarity to true stenocardia." Shall we not then agree with Dr. James Mackenzie that "*Pseudo-angina pectoris is a useless and misleading term*; *and it is time it was dropped out of medical literature*"[3] (author's own italics)?

Some writers thrust these counterfeits upon angina because of alleged intermediate instances. Now, although I have said that by intermediate cases one may step from any one disease of our nosology to any other, yet it is contrary to experience, at any rate to mine, to say that between these nervous storms and angina pectoris transitional cases are frequent and equivocal. The pain is different, the pulse is different, the panting is different, the behaviour is different, the storm is different, the duration is different, the causes are different, the issue is different.

This case, I suppose, would be styled transitional or ambiguous; of a lady of middle age, whose case a short time before had been diagnosed by an eminent London physician as angina pectoris. As he found her to be the subject also of "fatty heart," I assume that he regarded the case as angina in the proper sense, or at least as one of the "dilemma" cases. But, from an angina point of view, I was surprised to find that an intense orthopnea

[1] Ed. 1905, ii. 325.
[2] Gallavardin, *Mal. du cœur*, 1908.
[3] Mackenzie, Jas., *Heart Diseases*, p. 52.

was a part of the attacks, and one of these fortunately took place during my visit. Had my distinguished friend realised this feature, which presumably he did not witness, I think he would have hesitated in his opinion; for although a "fatty heart" may be curiously negative as to its signs (see p. 46, etc.), with such dyspnea other evidences of cardiac failure should have become manifest. But there was no lung crepitation, no tenderness of liver, no cyanosis, no fulness of cervical veins; and, save for some acceleration, the cardiac sounds and rhythm were normal. As the patient in her respiratory strife seemed to pay no heed to us, I said in a low voice to my colleague how odd it was that the alæ nasi were not distended. The nurse, of whom afterwards we made some further enquiries in another room, dropped the remark that as we left the bedroom the patient had asked her what was meant by the alæ nasi? On our return the alæ nasi were working actively! We plumped for hysteria, and treated the case accordingly; recovery was rapid and complete, and in a few weeks the lady was shopping in London, on foot. But as another example of the more ambiguous kind of cases, I will allude to the following instance:

Male I saw in consultation on suspicion of angina pectoris. The patient, a man of calm temperament, had suffered pain in his left arm, and from time to time from certain faints or confusions of sense. We were able however to detect the tender points usual in brachial neuritis, and the reactions in him were peculiarly ready, for to press on a certain point of the hand "made him feel queer," and he begged me to refrain, for a sharp and unexpected pressure there had "more than once made him faint right off." But I will not digress into a fuller story of this very interesting case of brachial neuritis with a good deal of vasomotor disturbance, which in some respects simulated angina. Of angina there was no evidence.

But I will not multiply illustrations of the vasomotor concomitants of irritation of sensory tracts, for almost any day we may find cases upon which to test and study them.

It seems probable that in all such cases the first vascular oscillation, however transient it may be, is constrictive, with a relative rise of central pressure; although in some a dilatation with falling pressures quickly becomes dominant. Even in typhoid, or other visceral, perforation it is stated that a

transitory rise of pressure precedes the substantial fall; and in chronic diarrheas, such as sprue, just before a large evacuation the pressure is found to rise sharply for the moment, and after the discharge to fall below the mean. And when constrictions occur suddenly over large areas, and are far more persistent, however free the organs may be from static disease, this exaltation of pressures often causes intense discomfort; and, as pressures vacillate, faintnesses, oppressions, pantings even to orthopnea, throbbings, palpitations, flushes, and other agitations, appear in sundry degrees and combinations. Intercostal neuralgia occurs usually in women, or fragile men; and in them, as the attacks are multiplied, vasomotor stability is more and more readily broken down. But, so long as heart and blood vessels preserve their normal structure, these faintnesses, palpitations, gaspings, stiflings, bodily agitations, hyperæsthesias, and psychical commotions are as wholly unlike the ruthless grip of angina pectoris as their frantic alarms are unlike its silent passion.

It is curious, as in the case last quoted, to see how often in these patients pressure upon the tender spots will excite or exacerbate the vasomotory seizures. The general condition of such patients also, the story of the case, and the tests of Weir Mitchell's or other "antineurotic" treatment, confirm a diagnosis which should very rarely present any real difficulty. Hasebroek's cases [1] of so-called "Angina pectoris," cured by such methods, were assuredly no more than phantoms of this sort. Let me reiterate that it is notable in the spurious attacks how obviously the heart is upset; we note palpitations, arrhythmias, sudden stoppages and accelerations, and so on, which are not characters of angina pectoris. In the neurotic cases the labile medullary centres seem to act more as an aggregate; respiratory, cardiac, and vasomotor balances vacillating more in unison, as they do in the ailments of children, whose centres are less stable and independent. It is remarkable that, in the bulbar area, the intense afference of angina seems more contained, especially if the attacks be infrequent; for in many cases vasomotor sympathy, far from being ascendant, is insignificant. In angina, as in migraine, a dis-

[1] Hasebroek, *Deutsche Arch. f. klin. Med.* Bd. lxxxvi.

order often classed on superficial analogy with angina,[1] the secondary vasomotor phenomena, if afterwards expansive, are also at first constrictive; so that in angina some areas of the surface of the body—especially the face and left arm—turn pale. We shall see that although generally in angina the face turns pallid, and the radial artery shrinks, this is not always so; the face may be flushed from the outset, and the artery expanded (*vide* p. 341). As however I have surmised already, even in these cases of migraine and angina some transient moment of constriction may precede, but so transiently as to escape observation. Indeed in this way vasoconstriction often becomes an active part of angina: a movement or an emotion raises arterial pressure, by this the sore parts are annoyed and, the medullary centres being irritated, pressures may rise more and more, and a vicious circle be established until by nitrites pressures are reduced, or by morphia the centres blocked.

On the other hand, no waves of constriction, not even the vasomotor convulsion of ague, will call forth angina in a patient free from the local lesion proper to it.

Such then are the neuralgic, neurotic, and vaso-vagal symptoms which, often engaging in outbreaks of their own, may occur also as secondary, or by-symptoms, of angina pectoris, as of any other irritation; but to exalt them to the rank of essential characters of this disease is a vagary avoided by the earlier observers, who knew them well enough. Yet in recent times, so widely have these superficial features imposed themselves upon teachers and taught, that when a year ago in a certain examination candidates were asked to describe angina pectoris, nearly all of them covered sheets of paper with descriptions of its spurious mimicries; some of them, indeed, losing themselves so far in the fog as to avoid the material part of the answer. And can we wonder at it when one of their distinguished teachers writes as follows: "Nothnagel established the fact (!)[2] that the characteristic phenomena of the condition are capable of being produced by functional conditions as well as by organic changes. It is

[1] The link, if any, between migraine and angina is atherosclerosis, which in the migrainous is often premature.

[2] The writer probably meant a conclusion, general statement, principle, maxim, or law, inferred from alleged facts. To take opinions for facts has not yet ceased to be one of the banes of science.

now universally admitted that angina may be true or false," *i.e.* that the case may be angina—always a menacing, too often a mortal disease—or may be something else, and trivial! As Artemus Ward said of the gorillas, "they are supposed to be human bein's to some extent, though they are not allowed to have a vote."

However this random thinking may now be left without further comment; though the false implications in the description of angina pectoris as the "graver variety of the disease" are so misleading as to be hard to excuse. The pale horse and his rider are not of these phantoms. How are we to complain that students never learn to think when, too often, they are exercised in analysis as slight as this? What, I repeat, should we think of a lecturer who threw together into the same class and under the same name the functional hemiplegias of hysteria and the hemiplegias due to cerebral disease! yet this is no forced comparison. Let us then hear less of pseudo-angina and more of pseudo-diagnosis. For happily we have many teachers more clear-headed; such as Edgren, who says plainly that he regards most at any rate of Nothnagel's cases of vasomotor angina as spurious; and Neuburger of Frankfort,[1] who, speaking from the vantage of an extraordinary experience (143 cases with 38 necropsies, most of them by Weigert), warns us against mistaking for functional nervous disturbances a malady which "always rests on an organic basis"; and Dr. Norman Moore, who says:[2] "Angina pectoris is generally associated with disease of the aortic valve; it is always associated with some degeneration of the heart." If in this last clause we shall find matter for controversy, the soundness of his clinical judgment amid so much froth is reassuring.

Certain sagacious physicians, who can see that "crises des nerfs" are a ghastly masquerade for angina pectoris, yet who are deceived by some superficial resemblances in these several processes, have surmised that the "real disease may arise out of the false one." Now, in the first place, we must not forget that in some cases of angina pectoris, especially in women, the agony of severe and repeated attacks may drive the sufferer into hysterical

[1] Neuburger, *Deutsche med. Wochenschr.*, Juni 13, 1901.
[2] Moore, Norman, *Clin. Journ.*, Oct. 19, 1907.

distress. So it was with the housemaid, under Dr. Hawkins and myself (p. 189), whose attack of chronic aortitis was so severe that the poor girl was driven to distraction. She became wayward: at one time unruly, passionate, and rebellious; at another, tearful and melancholy; and small blame to her. But her case, both in its active and ultimate stages, was far too unequivocal to permit us to suppose that the disease, whose agonies had put her thus beside herself, consisted in these scenes of shattered nerves. And there are certain far more unhappy illustrations of such an error, as when in a recent essay on the subject it was apparent to me that in a collection of cases, mostly of "pseudo-angina," the author had included two which were clearly cases of minor angina pectoris (so-called "stenocardia"). Yet the whole collection was classed as "hysteria" or "neurosis," and the author seems scarcely to have been taken aback by the sudden death of one of his two patients, for imperturbably he set down this incident as "secondary." Even among Sir William Osler's cases of "pseudo-angina," I suspect more than one was the genuine disease.

I have said that for a constructive review of "vaso-vagal disorders" we have the advantage of Sir William Gowers' perspicuous and practical essay; but in enquiring if the false may issue in the true, we must discern how far these several series of phenomena may be incidentally associated. In my experience, there have been two chief groups of these cases, the syncopic and the agitative; in the former group we may guess that dilatation in the splanchnic and other large areas is predominant, in the latter, constriction. And of such oscillations are those Secondary and Accessory Vasomotor Symptoms of angina pectoris (p. 226), symptoms which it has in common with other kinds of paroxysmal pain, yet which are its epiphenomena, not of its essential nature. And we shall admit that accessory vasomotor symptoms, if common epiphenomena of many irritations of large afferent nerves, and if standing in no direct affinity to angina, yet often play a considerable part in determining the moments of its assaults. Although then the accessory and the secondary processes may be in nature alike, or even identical, it is a part of a careful diagnosis to discriminate the Accessory symptoms—often accessory before the fact—from the

Secondary, or fringe, symptoms. For instance, as vasomotor oscillations may, as we have seen, be a product of angina—as of migraine, or other painful paroxysm, and so in comparison with the angina itself such riders be negligible; yet if, under an emotion, or a chill, or other such cause—as in a case of Broadbent's the vasoconstriction of a malarial seizure—this oscillation precede the anginal attack, by raising intra-aortic pressure it may stand in immediate causal relation to it. And often such vasomotor causes are as efficient as is muscular effort; so that the patient, whose attack to-day is determined by walking up an incline, to-morrow may suffer by the draught of a cold wind upon his skin, by a contact with cold bed-sheets, by the cutaneous vasoconstriction of digestion, by the influence of good or bad news, or some other annoyance. But to confound these proximate causes of an attack of the disease with the disease itself is not only an error, but a dangerous error.

Thus however it is that some discerning physicians still cling to the idea of an affinity between vaso-vagal attacks and angina; they fear lest the one, alien, even trivial, as in the first instance it may have been, may end, notwithstanding, in the other and graver malady. Even Balfour was tempted to regard false angina as a sort of immature or preliminary form of angina pectoris. In my experience I have never found reason to suspect, nor facts to suggest, any such concatenation. The cases of angina I have seen in younger persons have all originated in the specific infections—especially in syphilis, rheumatic fever, and influenza; atheroma pertains to later life. But of atheroma it may be surmised that in a life of continual vasomotor instability, due to turmoil or stress of mind, the aorta or heart may in time become strained; or that such an instability prevalent in certain persons, however easy their circumstances, may lead at length to cardiovascular deterioration. Yes; such a chain of causes is easily imagined; but, before it can be taken seriously, we must have more evidence of it than at present we possess. The difference is not one of issues only, but also of processes. In 1877 I wrote a paper to suggest that "granular kidney" may arise from mental vexation over long periods of years, and the opinion has since received general acceptation, perhaps substantial proof; but my notion was, and is, that such

stresses act rather by obscure perversions of metabolism than by the continual labouring of the arteries by incessant vasomotor or vacillatory tides. So in the essay on Arteriosclerosis in this book, I have discussed mental anxiety as a cause of Hyperpiesia without renal disease; now hyperpiesia may be no doubt a direct cause of atheroma, and so a contributory cause both of angina, as a disease, and, incidentally, of its several attacks; but this long insidious process is scarcely what is meant by the teachers to whom I refer. Their eyes are fixed, as their descriptions declare, upon the more or less immediate transition of some of the multiform vasomotor storms of the kind and character of "pseudo-angina"—I say "multiform," for the descriptions of various observers, as for example of Nothnagel, of Douglas Powell, of Gowers, or of myself, differ widely—into another kind, the kind of angina pectoris; the inference being that those storms ought therefore to be interpreted as potential angina, the one being prone to merge into the other. This is the opinion which I regard as erroneous and deceptive. All the alleged cases of conversion of pseudo-angina into genuine angina which I have seen, or read of, were a development, not of a "true" out of a "false" angina, but of a true out of a false diagnosis; the cases, if at any time angina, were of this nature from the beginning (*vide* p. 241). At the same time I do not contest the opinion of Sir Richard Douglas Powell, whose experience in these maladies is very large, that in finance, politics, and other spheres of intense nervous strain, arterial tension may range high, and be favourable to arterial lesions; and that, prior to angina, such vexations may favour severe vasomotor oscillations, such as to undermine the blood vessels. This is no unreasonable speculation, but it is concerned surely, not with vasomotor angina, which does not beset such persons, but with the engendering of angina proper by way of subverted nutrition and atherosclerosis.[1] For my own part, of the scores and scores of cases of pseudo-angina which I have seen, or of which I have heard or read, I cannot remember one which ended in angina proper.

The pains and distresses consequent upon too liberal doses of thyroid extract in myxœdema have been compared to angina.

[1] For an analysis of these subtler causes see also Dr. Mitchell Bruce's *Lumleian Lectures* for 1911, and Vol. I. p. 236 of this work.

Suffocative feelings and pantings there are, but the pains are widespread, and occupy arms, legs, and trunk. One such case I recall vividly; the pain and distress were bad enough, but a comparison with angina could only be fanciful.

But to return from such speculations, full of interest as they may be, and to emphasise the responsibility which lies upon us all to beware of the grave, and possibly calamitous, error of mistaking angina pectoris for a nervous squall, I will illustrate this error again by brief allusions to three, out of more cases on my notes, in which it was not avoided; and also to one recorded by Sir James Goodhart. I will take them from the list of women, as with them it is that we are more apt to be led astray.

Mrs. C. was a very highly strung woman, full of life and social accomplishments. Her sister had suffered for two or three years from vaso-vagal attacks and irregularities of the heart, but she herself had enjoyed excellent health. After the menopause she got too fat, though her energy and vivacity suffered little diminution. Keeping a generous table for her friends she probably lived, from a physiological point of view, rather too well. When about 55 years of age she began to suffer from "liver," and from "oppressions," especially on walking up inclines. In a woman of her temperament and offhand, unselfish way of describing her symptoms, what more natural than to attribute them to "pseudo-angina"; and this the clearer as her nerves had been shaken by more than one heavy trial. And such was the opinion of more than one physician. But at length this arrest or pain at mid and upper sternum made it almost impossible for her to walk far uphill; the pain was not in itself severe, but she was aware of an imperious arrest and an indescribable warning. I found her arterial pressures very high, the pulse sustained, and the heart's apex an inch or more out to the left. On slight exertion a faint systolic murmur was brought out at the base of the heart; and the second sound was noisy, right up to the apex. The aortic dulness extended 3 cm. to the right, and the vessel in the suprasternal notch thumped against the finger. The urine, repeatedly examined, was of full value, and contained neither albumen nor casts.

Her case thus proved to be no "pseudo" anything, but a very real something; namely, angina minor and hyperpiesia. For two years we kept the malady at bay, and happily the angina never rose to the major form; indeed, as her state deteriorated, which was by way of cardiac defeat under the high pressures,

the oppression abated; and before her death, in the ordinary course of cardiac dilatation, it had disappeared.

The second case was in a lady, Mrs. K., of similar social position, but of placid temper, and, on the whole, fortunate in the events of her life. She likewise, about the same age, found herself checked by a substernal compression on walking uphill, but was assured again and again that her case was a "pseudo-angina." Her symptoms however, when I saw her, were in site and kind like those of Mrs. C., but her blood pressures were more amenable to treatment. For various periods she was under Dr. Roberts of Harrogate, who kept long charts of her blood pressures under several methods of treatment. In her case a course of high frequency currents certainly seemed, more than once, to effect an abiding reduction of the arterial pressures, so that the symptoms were alleviated. Thus by one method or another, of which a rigid diet was always a part, the angina minor disappeared for a while, and for two or three years she remained fairly well; but the angina returned, at first in the same minor form when ascending hills, but later in a severe and classical form, especially in the left arm. She died in an attack. (See p. 110.)

It is surprising how long this minor form, especially in hyperpiesia, may dog a patient without advancing to a fatal stage.

The third case was of a lady also in later middle life, left by the death of her husband with the anxious charges of a young family and a large estate. Her case was one of those I cited in my paper to the Hunterian Society in January 1895. Though a woman of great parts and energy, she could not be called neurotic in any morbid sense; but she was perhaps gouty in person, certainly she came of a gouty stock. She, likewise, was "pulled up" one day on walking up hill by angina minor, and likewise was assured it was "angina spuria." I saw her after she had felt repeated arrests of the kind, and found her arterial pressures too high. There were no signs of renal disease. For a long time diet, nitrites, and other appropriate means were successful in warding off, mitigating, and perhaps arresting the hyperpiesia and angina; but, unhappily, before many more years had passed, she succumbed to a rapidly growing uterine carcinoma.

These are three instances, out of many which I could cite from my own records, of angina minor, a malady far from uncommon, which were interpreted as "pseudo-angina."

The next illustration of the danger of thus paltering with

diagnosis in matters so serious, I have taken from a lecture by Sir James Goodhart :[1]

Male, æt. 60, consulted " a physician in town, who said he was suffering from pseudo-angina." At a later date he saw another well-known physician, who also, I suspect from motives of humanity, called it a " false angina." But the case proved then and afterwards to be only too genuine.

And another reported by Dr. Core :

Female, æt. 48. Recurrent attacks of intense pain in the chest radiating down the left arm ; considered to be " pseudo-angina." However arteries " stiff " and aortic second sound reinforced. Systolic aortic murmur. No history of rheumatism. Blood pressures during stay in hospital 190-200. A few months after discharge died of cerebral hæmorrhage. (This case was probably not syphilitic ; in syphilis the arterial pressures are usually moderate, and death not by ordinary cerebral hæmorrhage.—C. A.)

Bureau[2] well says : "Above all it is when aortitis occurs in a youngish woman that the mistake may be made " (of regarding it as mock angina). He quotes a case from Bucquoy,[3] in which, after some hours of distress interpreted as " globus," the patient suddenly had a typical seizure of angina pectoris and died in it.

Humanitarian motives do not justify deliberate deception ; however I suspect that these sadly erroneous opinions were given in good faith, and fell in with the fashion of confusing angina pectoris with nervous squalls. But this kind of plausible or irresolute diagnosis is doing more harm than we realise.

Of angina pectoris as an alleged event of Graves' disease I know nothing. I have seen nothing more than oppression with palpitation, " point de coté," and pains about the chest and arms not characteristic of angina proper.

[1] " On Angina Pectoris," *Clin. Journ.*, April 4, 1894.

[2] Bureau, *Aortites aiguës*, Thèse de Paris, 1893.

[3] Bucquoy (quoted Bureau), " Clin. sur l'aortite aiguë," *Journ. de méd. et de chirurg. prat.*, 1882.

CHAPTER III

TOBACCO ANGINA

Tobacco Angina, if this name is to be received, is a curious and rather rare affection. It was recognised first, I think, by Beau,[1] who described eight cases. I have seen only three well-marked cases: in two of these the attacks were mild; in the third they were of intense violence. But even this patient had scarcely the aspect of angina pectoris; it was not a state of awe-stricken arrest, but a strife. The patient, a strong man of middle life, but presenting some signs of arteriosclerosis, had an attack in my presence: he writhed in a passion of distress; by his wrestling with the invisible foe the sofa on which he was lying was partly overturned, so that from the back of it he rolled on the floor. It was difficult to get him to say exactly, even after the attack, where the pain was, or if it were pain, or some other distress, which made him frantic; but it seemed certainly to be a thoracic, and apparently a cardiac oppression, and, taken alone, reminded me of the agony of a clot in the heart. There was no more dyspnea than the muscular effort would naturally produce; there was physical alarm, but perhaps not the peculiar angor animi. The attack lasted some three or four minutes. To note the behaviour of the heart during the height of the seizure was impossible; but as it declined, the action was rapid and irregular. Moreover in the tranquil intervals more or less arrhythmia continued, an arrhythmia having the common characters of tobacco heart. The well-marked premature systoles, the palpitation, the agitation, and the audible characters of the heart, suggested

[1] Beau, *Arch. gén. de méd.*, 1862.

tobacco; so that I took upon me, too confidently perhaps, to assure the patient, who had some knowledge of medicine, that his fears of angina pectoris were unfounded. He was a very persistent smoker of strong cigars, and on restriction of this habit the symptoms abated.

Cases of tobacco-poisoning which, if in the intermittent returns and the kind of distress they fall far short of such anginoid attacks as that just described, yet have somewhat similar features, are not very uncommon. For a fuller description of these cases, which may simulate degrees of angina pectoris, I may refer the reader to the section on Tobacco Heart in my chapter on "Functional Diseases of the Heart" in Allbutt and Rolleston's *System*. But here I may dwell for a moment on these lighter simulations, for their signs may be more equivocal than those of the violent seizures. Indeed in the minor cases there is not rarely a sternal or "substernal" oppression such as to arrest or deter the patient as he ascends a hill, or at least to dispose him to stop. Many years ago a medical friend of mine, now in excellent health, but who had then been smoking too many cigarettes, told me that in walking up a hill a tight, constricting pain would catch him across the mid-chest. If he stopped it passed off. The tobacco was discarded, and with it the pain departed. But, on reflection, and speaking as a physician, he said he could not have distinguished the pain from a slight angina pectoris. Dr. Mitchell Bruce says that in tobacco cases the pain occasionally passes into the left arm; this I have not observed in cases wholly free from suspicion of true angina. (Dr. Bruce lays no stress on tobacco as a cause of arteriosclerosis.) In a few moments, often with some intermittence of the heart and an eructation of wind, the attack passes away; but this it may do in true angina. As the attacks may very suddenly appear, so when the poison is laid aside they soon vanish; still, in a man of middle age with arteries no longer at their best, the diagnosis might for a time have to be held in suspense. Dr. Bruce therefore, while warning us lest we pronounce a case, which is but tobacco-poisoning, to be genuine angina pectoris, quotes a case in a man, æt. 60, who, after some ominous stenocardia—such as I have described above—had a "fearful pain of the chest, and down the left arm,

cold sweats, and a sense that his last hour had come" ; yet on dropping his tobacco he recovered. It is not quite clear that in this elderly man the case did not partake of the nature of genuine angina, attacks of which may have been determined in an atheromatous aorta by the vasoconstrictive effects of tobacco on the intra-aortic pressures. Or, if it be true, as alleged, that the use of tobacco leads to arterial disease (*vide* Arteriosclerosis, Vol. I. p. 250), it may provoke aortic lesions out of which angina pectoris may arise. In a gentleman, æt. 53, who called upon me not long ago not for angina but for cardiac oppression, arrhythmia, and palpitation due to tobacco, I found the systolic pressure was fully 160. No peripheral atheroma was palpable.

The genuine spectral horror or awe of angina I have never observed in mere tobacco angina, but reasonable alarm only. The vertigo, so frequent in tobacco excess, is often in part a cause of the alarm. Dr. Lloyd Jones of Cambridge kindly permits me to say that he has had not a few attacks of tobacco angina, attacks which at first he supposed to belong to the genuine disease. Some of the attacks were severe but, after no little anxiety in respect of them, he became convinced that they were due to increases of his generally moderate allowance of tobacco ; as for instance during a holiday or on a long railway journey. Dr. Jones thinks that in his case the attacks were attended with sharp rises of blood pressure ; at any rate they were always promptly relieved by a whiff of amyl nitrite.

But some of these tobacco cases simulate rather the false than the genuine angina.

Male, age about 45, brought to me by Dr. Poignand of Walsham le Willows, smoked strong cigars all day, and indulged freely also in food and drink. He had become subject to " syncopic attacks " of long and alarming duration. A persistent, not paroxysmal, aching pain was seated in the left chest between the third and the seventh ribs. The pain was independent of exertion. In the definite seizures the pulse disappeared ; near them, or in less severe attacks, it would run up to 160. By strict obedience to our directions the patient completely recovered ; but four months elapsed before any definite amendment could be relied upon.

It so happened that, after writing these words, a gentleman, æt. 40, who had suffered from attacks of this kind, called

upon me again for an opinion. He had dropped his tobacco (forty cigarettes a day!) for just four months, but still was complaining a little. However, except an occasional extra-systole, I could find nothing wrong with heart, arterial tree, or blood pressures.

Edgren records two cases of severe tobacco angina, and says that they are very different (*sehr verschieden*) from the genuine disease. And perhaps some cases by other writers, recorded as "vasomotor angina" and so forth, may be recognised by the wary reader as pertaining to tobacco. That wearisome importunate submammary ache, not anginous, is often notable in tobacco cases. Von Basch witnessed, and promptly diagnosed, one severe case of the kind in a woman; in women, at any rate in England, I am thankful to say, this cause might well escape our suspicion. An arrhythmia, independent of the attacks, may serve as a note of distinction; and, what is rare in angina pectoris, the patient will complain of this cardiac irregularity and palpitation independently of the attacks.

Huchard asserted that tobacco angina was caused by constriction of the coronary arteries, being, as it were, a sketch of genuine angina; a postulate which, to less adventurous thinkers, seems to be a mere guess, and an unlikely one; for, as we shall see subsequently (p. 356), it is inconsistent with what is known of the physiology of these vessels. A kind of "pseudo-angina" it may be, probably is.

A case commented upon by Mouriquand and Bouchat [1] well illustrates the occasional difficulty of detecting the true nature of particular cases:

Male, æt. 54. Sudden seizure of angina pectoris. Previous health good. No alcohol. No rheumatism. Syphilis denied, and no sign of it found. But a very heavy smoker—thirty to forty cigarettes a day as well as several cigars. Cardiac signs practically negative. *P.M.*—Heart practically normal, aortic valve healthy. On ascending aorta several gelatiniform patches. Orifices of coronary arteries obliterated by such patches, but orifice of right coronary artery just visible.

Now, as on microscopic examination these patches proved to be a certain typical subinflammatory disease of the vessel,

[1] Mouriquand and Bouchat, *Arch. des mal. du cœur*, oct. 12, 1912.

especially of the media, and to differ from the degenerative characters of arteriosclerosis, they unhesitatingly and no doubt truly recorded the case as syphilitic, and not due to tobacco. The authors consider that the attribution of genuine angina to tobacco is unproven. I also believe that many cases of true angina, infective cases for example ending in recovery, are erroneously put down to tobacco.

CHAPTER IV

CAUSES OF ANGINA PECTORIS

A. Remoter Causes.—*Age.*—Angina is by no means unknown in the aged and decayed, but its greater prevalence is at a somewhat earlier stage, in the years of senescence; in senectus, not in senium. However, no general rule can for the present be accepted, as our statistics are vitiated by arbitrary or promiscuous collection of cases. Angina in its typical manifestations is far from unknown in young persons. Heberden refers to a case in a boy of 12 (due, probably, to rheumatic fever or other infection) (see Aortitis, p. 150); his "Unknown" was only æt. 46 (syphilis?). Fothergill publishes two typical cases beginning at the ages of 25 and 30 respectively (probably syphilitic). One patient died, the other recovered. He comments on this recovery as in his experience unique. One of Allan Burns' patients suffered from angina during the age period 36 to 40. He died of cardiac dropsy, and probably the whole morbid series was syphilitic. I have notes of at least three cases in adolescence—two in senior schoolboys, one in an undergraduate; all three were cases of rheumatic fever with aortic disease, and all, so far as the angina was concerned, ended in recovery. I have seen also two definite cases in children, the younger one a case of rheumatic pericarditis (also with recovery); and the late Dr. Gibson showed before the Medico-Chirurgical Society, in March 1903, a child, the subject of angina and aortic valvular disease of rheumatic origin. Dr. Hugh Stewart[1] has published a case of angina pectoris (with aortic and other valvular disease) in a boy æt. 7; the angina

[1] Stewart, Hugh, *Edinburgh Medical Journal*, June 1906.

came on towards the conclusion of a severe rheumatic fever—first indefinitely, then in frequent and "typical attacks." There was no cyanosis, no dyspnea; but sudden pallor, pain, arrest, and anxiety. Such was the older child of my two, a girl æt. 12½, whom I saw in private consultation. At the end of an attack of rheumatic fever she also complained of intense retrosternal pain and constriction, the pain radiating in the usual way down the left arm. During a partial recovery she died suddenly in an attack. Unfortunately, no necropsy could be obtained. These cases in young persons with sound hearts generally end in recovery, so far as the angina is concerned; and thus they fail to appear as such in the Registrar-General's Reports; so it is also with many of the syphilitic and most of the influenzal cases.

The many syphilitic cases fall for the most part between the ages of 40 and 50; but not very rarely between 25 and 35. Sir William Osler[1] publishes a case in a man æt. 24, whose primary infection was nine years before. Neuburger (*loc. cit.*) includes two cases (in males) of æt. 31 and 37. In middle life cases of angina largely increase in number, the period of greatest frequency being from æt. 55-65. Seneca's first attack was at the age of 60. I have notes of two patients in extreme old age, viz. 83 and 90 respectively; the second case is mentioned p. 514.

Osler, of 40 cases, had 4 under æt. 40; 13 between æt. 40 and 50, 13 between æt. 60 and 70, and 9 between 70 and 80. He gives the age periods for larger numbers as follows: Under 30 years, 9 cases; 30-40 years, 41 cases; 40-50 years, 59 cases; 50-60 years, 81 cases; 60-70 years, 62 cases; 70-80 years, 13 cases; above the age of 80, 3 cases.

Station of Life.—It may be true, on the whole, that the disease falls rather upon the upper classes, but I have not found it so rare in hospitals as is commonly supposed; such a distribution of the disease may be apparent only, and governed by the chances of individual practice, the prevalence of syphilis, and so on. The same remark is made concerning gout, that it is a disease of the easy and intellectual classes. Now both gout and angina not infrequently, though by no means always in either case (Vol. I. p. 273), are associated with high arterial pressures; and the persons

[1] Osler, W., *Lumleian Lectures.*

of whom we are speaking, as a class are sedentary and consume larger quantities of the richer foods. However we must first be sure of our facts, for Samberger,[1] of 73 cases in the Policlinic of Prague (1895–98), says " all these patients belonged to the class of poor working people."

Sex.—The comparative rarity in women seems to be unquestionable, although statistical reports scarcely bear out an extreme disproportion (see also section Aortitis, p. 172). Heberden thinks he saw in all about 100 cases, of which only 3 were in women. In Husband's 237 collected cases women were in the proportion of 1 to 60. Sir John Forbes, for his *Cyclopædia of Practical Medicine*, collected 88 cases, of which 8 were in women. Osler in his 40 cases saw 1 woman only; these fractions, to judge by my own experience, are too small. Consultations on behalf of heads of families are more frequent, and in the case of men of distinction are demanded also in respect of the public. Burwinkel,[2] in a collection of 117 cases, found the women to the men as 1 : 7; of Neuburger's own 143 cases 118 were male, 25 female. I have seen many cases in women; I recall at once, as I write, ten or a dozen of the most typical in kind and degree. Certainly angina minor (" stenocardia "), often met with in the course of Hyperpiesia, is by no means very rare in women. So of Samberger's 73 cases, about one-third were in women. Of this difference of sexual susceptibility no very definite account has been given, for women also have coronary arteries; it may be surmised that the causes lie, in some measure at least, in the smaller incidence of syphilis and gout on women, and perhaps in the protection of the thoracic aorta from physical strain. Were angina pectoris " of the nature of neurosis," it would on the contrary be more prevalent in women. The neurotic crises confused with angina, a disease with which, as we have seen, they have nothing in common, neither in nature nor symptoms, do fall chiefly upon women, and feminine men.

Heredity is of course not a " Cause "; paradoxically it might be called the absence of causation, an apparent severance of

[1] Samberger, *Bohemian Arch. of Clin. Medicine*, vol. i. fasc. i. 1899, quoted *Epit. Brit. Med. Journ.*, June 17, 1899.

[2] Burwinkel, *Deutsche med. Wochenschr.*, April 5, 1906.

the chain of causation; but the detachment of the individual from the stem is no very radical schism in a continuous growth. Heredity is then the cause of a quality in the offspring only after the manner in which the finger may be regarded as the cause of the finger-nail. Moreover there is too much contingency in its causation (infections and habits) to make transmission apparent. Nevertheless a proclivity to angina may run in families; such examples are not infrequent. I think Robert Hamilton [1] was the first to notice this reappearance. Neuburger, in 20 of his 143 cases, found a history of angina in blood kindred. It is often stated that it is usual in angina pectoris to find a hereditary tendency to irritability of the nervous system. This notion, which in my opinion is baseless, has probably arisen out of erroneous diagnosis.

Season.—Heberden concluded that angina has no periods of seasonal prevalence, and subsequent teachers have agreed with him.

B. Proximate Causes.—Whatsoever the nature and seat of angina pectoris, I must insist that its causes have much in common with those which determine arterial disease generally. These causes I have set forth so far at length in my essays on Arteriosclerosis and Aortitis, that here I have only to indicate their special bearing upon this peculiar appanage of arterial disease; and for this purpose we may consider five chiefly: (1) the strain of bodily labour; (2) the strain of high blood pressures; (3) mental and emotional causes; (4) infections; (5) some of the obscurer causes of atherosclerosis.

1. *Bodily labour*, so far as we can discriminate its effects, seems, taken alone, to be no very fruitful parent of angina. Though not a few cases are recorded in forgemen and other Anakim, in many of whom probably the angina was syphilitic, for it was often associated with aneurysm (*vide* p. 188), Sir Richard Douglas Powell hesitates to regard physical stresses as frequent causes of angina; and Dr. Colbeck concludes that, infections apart, it is far from common in blacksmiths, navvies, and other such labourers (see however case of Bartholmes, p. 366). Gibson, on the contrary, did "not doubt that many of those whose avocations lead them to physical overstress

[1] Hamilton, R., *Med. Comm.*, 1780, vol. ix. p. 307.

are liable to angina pectoris"; and he added that in his experience of the prevalence of the disease in the hospitals of "districts where hard mechanical occupation is the rule, angina pectoris is far from uncommon amongst working people."[1] This important opinion may need the qualification that, like general paralysis, this prevalence among labourers is urban rather than rural; it prevails where syphilis prevails. In my experience, infectious cases, as of rheumatism or syphilis, omitted, its incidence upon the labouring classes in Leeds was not remarkably high. Indeed in my own notes I find but one case in which labour may have been the chief cause; and, as the patient was a soldier, it is not unlikely that there also syphilis entered into the causation.

T. B., æt. 26, had been exposed on service to exceptionally heavy labour. He was the subject of severe angina pectoris in its classical form, including dread. He answered questions frankly, but neither in the history nor present signs were traces of rheumatic fever or of syphilis to be verified. The first attack was certainly produced immediately under severe bodily effort; and since this event he had been liable to seizures on slighter efforts, so that he was unable to follow even light employments. Wa.R. not tried.

A similar causation was supposed in the young housemaid under Dr. Hawkins and myself (p. 189).

It is surprising indeed that not more cases of angina are attributable to single violent efforts. If such were the case with T. B. and the housemaid, yet in the vast majority of recorded cases, if the moment of the first attack were determined by effort it was by an effort comparatively slight—such as an ascent of no very steep acclivity, a walk against a wind, or the wielding of a weapon no more formidable than a fishing-rod or golf club. And in many of these cases, if the first declaration of the evil were thus called forth, on close enquiry it proved that the crisis had been preceded by some warnings; as by transient oppression in the form of angina minor—a momentary tightness under the breast bone, or a vague and transitory sense of arrest—sensations obscurely ominous of what was incubating within. One gentleman indeed, a resident in Halifax, did tell me his first attack was as a bolt from the blue, while after a hard day's sport he was bicycling

[1] Gibson, G. A., *Diseases of Heart and Aorta*, p. 763.

home up a steep hill, in happy rivalry with his sons and daughters; and Latham, unaware of the infections, thought "some considerable effort" started the disease in the majority of his cases. When disease has corroded the intima and media, some rather greater effort produces a rise of pressure which the old coats no longer resist; it forces the media, and the strain, reaching the adventitia, tugs upon it as abdominal tensions stretch the mesentery (Vol. I. p. 447 and II. 194). Dr. Mackenzie[1] describes a case in a builder, æt. 48, in whom a first attack of angina reached its maximum two hours after a violent bodily strain. We know that an apparently normal aorta may split (see p. 206), and a split might not be immediately mortal; but it is more probable that, in such cases as that of Dr. Mackenzie, the strain took effect at a spot of pre-existing syphilitic or atheromatous disease. In one of Broadbent's cases (pp. 301, 302) the initiation of angina was traumatic; a severe crush of the chest gave rise to distressing attacks of angina and a double aortic murmur: we know that many an aneurysm has been thus determined, though syphilis is at work more frequently than we have been wont to suppose.

That the moments of the several seizures are often determined by effort, even by effort of mind or emotion, we know only too well; so well indeed that there is a notion abroad—one which more than once has been mentioned to me in discussing a diagnosis—that attacks are always so determined. Huchard countenanced this erroneous notion. Yet from Heberden himself we have learned that attacks may occur during sleep, often during rest. Diderot died of angina, and a very severe attack is recorded which awoke him from sleep. Such nocturnal accesses may be in obedience to secret tides of blood pressure;[2] and we shall remember that an apoplexy not infrequently occurs during sleep. In an old gentleman suffering from angina, since dead, whom I saw with Mr. Atkinson of Saffron Walden in the autumn of 1911, the attacks, which were many, had generally occurred between 2 and 3 A.M. A nocturnal attack, which may be quite as severe as at other

[1] Mackenzie, Jas., *Brit. Med. Journ.*, June 30, 1906.

[2] See Dr. Ewart, *Lancet*, Sept. 15, 1906, who suggests fatigue toxins; and Dr. Gordon of Exeter, *Lancet*, August 7, 1909.

hours, and is aggravated by its untimeliness, makes one point of diagnosis from mock angina; but tobacco seizures, whether anginoid or of cardiac disorder, do sometimes occur at ghostly hours. One such case I well remember.

2. *The Strain of continuously High Blood Pressure.*—It is not necessary then to assume that, for injury of the vessels about the heart, pressures must have run for some years continuously at excessive heights; though of course we might expect this to be no rare experience. In not a few persons, probably in the majority of cases, atheroma becomes established in the aorta-coronary area, not so much because the blood pressures had been positively excessive, as because these parts, whether by original structure or by the insidious influence of some unrecognised toxic or other morbid process, were vulnerable. However, the belief is prevalent that angina pectoris is correlated with excessive blood pressures. Although in Hyperpiesia it is true, as we should expect, that high pressures coinciding with vascular disease result in a larger proportion of anginal coincidence, yet even if we lump Hyperpiesia and Bright's disease together, we shall find angina mentioned in but a small minority of these two classes. Sir James Goodhart (*loc. cit.*) observes how commonly "the extremest arterial high tension may be quite free from pain." Given however a defective aorta, the intercurrence of high pressures is likely to promote angina, or to aggravate it. Moreover, angina is, for the greater part, a disease of the elderly, in whom pressures naturally rule a little higher. Notwithstanding, moderate pressures are quite consistent with angina, and are not infrequent in it, as Vaquez, and even Pal, have carefully noted in not a few cases;[1] Dr. Mackenzie, as we shall see, has verified this conclusion. For instance, in my many syphilitic cases I have found moderate pressures to be the rule. Kauffmann[2] says that pain (with the normal heart) raises the minute volume, the blood pressure, and the heart's work; thus the arterial pressures may be driven up only during the attack itself, to subside during the tranquil intervals. For obvious reasons the blood pressure can rarely be taken during a severe attack; but in a certain elderly lady two of us

[1] See, *e.g.*, *B. and M. Soc., Méd. des hôp.*, juin 28, 1906, p. 663.
[2] Kauffmann, *Zeitschr. f. exp. Pathol. u. Therap.* Bd. xii., April 24, 1913.

found the systolic pressures during attacks to be 166-180; in the intervals only 140. The difference may have been wholly emotional. Dr. Morison in one case (a man æt. 73) found the pressure during an attack to be 140-145 (pulse 84 mm.). Drs. Mackenzie and Price informed me, in a private letter, that (in a case of syphilitic aortitis verified post-mortem) the systolic pressure, ordinarily 120, rose during an attack to 140. I have notes of some families of which certain members were the subjects of hyperpiesia, others of angina pectoris. On the contrary, in another case of mine we found during an attack that the pressure fell to 120, indicating not so much vasodilatation as cardiac inhibition; for, after relief of the pain by antipyrine, the pressure rose again to 140. If however the aortic pressure falls low, anginal attacks may and usually do cease; though Huchard's assertion that "L'angine de poitrine ne peut plus se produire chez un malade en état d'hypotension artérielle" betrays his besetting incaution. An interesting example of this sequence is reported by Dr. Bruce Porter[1] in a case of angina pectoris with aortic disease. The patient was a man, æt. 43, in whom, during a gradual progress of heart failure, the attacks did not cease until the systolic pressures had fallen to about 100 mm. In another case, of Dr. Morison's (a man æt. 32), during the attack "a musical murmur" (of aortic regurgitation) was almost hushed; yet the attack was relieved by nitrites. Records during attacks are too scanty to justify any general conclusion, but we may take it that there is no direct relation between arterial pressures and angina.

3. *Mental and Emotional Causes.*—He is a happy man who in middle and later life can look back upon a many years without strife and disappointment; not a few patients of angina have no doubt been worn down by such trials. On the other hand, many happy and thriving country gentlemen, and successful professional men and tradesmen, suddenly seized by angina, have no grave record of the kind, nor look back upon any "neurotic" family history; so that if, as I have supposed under Arteriosclerosis, certain cases may have been engendered by such cares, these in my experience have not been widely efficient, nor formed a characteristic source of angina. The great stress laid by some

[1] Porter, Bruce, *Brit. Med. Journ.*, Nov. 21, 1904.

physicians upon business cares and excitements indicates the sphere of their practice among the wealthier commercial folk, who are prone to eat and drink too much. And may not such attributions as "domestic worries" and "infelicities" sometimes cover a secret syphilis? For other causes of atheroma, and so incidentally of angina, I may refer my readers to my essay on the causes of Arteriosclerosis (p. 148).

4. *Infections: Gout.*—Our gluttonous and bibulous ancestors, to judge by the records of their physicians, seem to have been subject to terrible conflicts which were lumped indiscriminately together under the conjectural titles of Gout at the Heart, at the Stomach, or at the Diaphragm (Butter). Fothergill held the gouty hypothesis, and Materia arthritica retropulsa was Hoffmann's explanatory title for Angina. Some of these cases seem to have been angina, others the dyspnea of uræmia or hyperpiesis, others turbulent cardiac arrhythmia, gastric fermentation and colic, and so forth. The description of such cases by Sydenham, by Heberden, and by many other authors in the eighteenth and early nineteenth centuries, are full of interesting matter. If we claim more discernment, we have not yet the data for an analysis and redistribution of the terms of all their series; but from general impressions we gather a substantial remnant of evidence in favour of some long association between gout and angina pectoris. Butter (*l.c.* p. 304), Elsner,[1] and Hoffmann convinced themselves of a causal bond between angina and gout, or some kind of "arthritism"; and, as Elsner was the first herald of angina in Germany, so this arthritic hypothesis of the disease gained a strong foothold in that country. Wichmann[2] however warned his countrymen that coincidence is not necessarily causation; he remarked that itch might occur in a syphilitic patient, yet stand in no causal relation to this cachexia (p. 192). In his not very important tract on Angina Butter supposes angina to be diaphragmatic gout; and Erasmus Darwin was of a like opinion. Wall said, "Angina et podagra alternatim per intervalla ægrum vexant"; Canstatt said the same; Forbes agreed, but put plethora—or rather gorging and tippling—in the first place, as contributing to a common sub-

[1] Elsner, *Abhandlung über Brustbraune*, Königsberg, 1778.
[2] Wichmann, *Ideen zur Diagnostik*, Hannover, 1797.

stratum of disease. "Plethora" was often in the mouths of our forefathers, who generally meant by it what recent writers mean by "high tension." At the present day, Ebstein, in his work on Gout (1906),[1] says, a little paradoxically, that angina pectoris is, as it were, an attack of gout; his meaning seems to be that angina pectoris is a play of this irritant humour upon the nerves of the heart, so that angina appears to him "lediglich auf nervöser Basis zu beruhen" (p. 256): on what nerves he does not state. In 194 cases of gout he found 72 with cardiac troubles, and 97 (!) with angina pectoris. He admits that in subjects of angina there is often no history of frank gout, but he reads many of them nevertheless as of gouty stock. More particularly, he says angina is apt to appear in the glycosuric gouty; but, as even Ebstein is foggy in his diagnosis of angina, and evidently was led astray more than once by cases of the spurious disease, and in other cases by paroxysmal dyspnea, we cannot lay much stress on his calculations. Certainly they are not borne out by ordinary experience. I cannot say that I have found angina more common in gouty glycosurics than in other elderly men with underlying arterial degradations. So far as I am aware, the most valuable personal observations on gout and aortic disease are contained in Dr. Mitchell Bruce's *Lettsomian* and *Lumleian Lectures* (1901 and 1911), to which again and again I have to acknowledge my debt. On this part of the subject he says,[2] "One-fourth of all cases of "gouty heart" have pains of anginal character, and often pronounced angina." In twelve out of twenty-nine of his cases of gouty heart a systolic murmur was heard in the aortic area, conducted into the carotids—a sign of deterioration of the aortic arch and valve. This statement confirms the opinions of Sir Dyce Duckworth, of Dr. Norman Moore, and indeed of Murchison, on the earlier incidence of disease of the arteries, especially in the aortic area, in persons actually gouty or of gouty habit (Vol. I. p. 273). As an extreme instance, Dr. Bruce relates the history of a patient, who before the age of 20 had suffered from gout, both articular and irregular, and in whom, at this precocious age, angina pectoris supervened. Could we have a stronger suggestion than is

[1] See also *Berl. klin. Wochenschr.*, June 24, 1895.
[2] Bruce, Mitchell, *Lancet*, May 23, 1901.

offered to us by this prevalence of angina in the ill-defined group of gouty persons, of the origin of angina, not in the heart—for the heart in these cases, some of which are cases of high arterial pressure, is, until strained or very atheromatous, if abnormal, abnormally strong—but in the aorta, and especially in that part of it which abuts upon its valve. If this atheroma occlude the coronary orifices, or so invade their trunks as to cut off the more direct blood-supply to the heart, making it thus less able to evade inhibition, then the angina becomes more than a painful malady, it becomes a treacherous menace to life. Among the more interesting records of the older physicians are those cases in which anginiform states gave way to podagrous seizures, and conversely. Dr. Gayford kindly sent me the notes of such a case, which ran as follows:

Male, æt. 68, engaged in large commercial affairs. In spring "could not shake off a cold," and frequently had recurrences of pain and tightness across the chest; these would pull him up while walking, and might seize him even while lying quietly in bed. The pain would extend to the left shoulder, and at times down the left arm. The heart at such times was most irregular, often 120, with a cantering rhythm. The pulse was not hard, and the radial artery was in fair condition. There was no murmur, but the heart's action was tumultuous. Trinitine relieved these states. At other times a rapid cardiac rate would set in, without pain—when the pulse would be small and soft. On November 21, "had an attack of heart failure; pulse uncountable, jugulars distended and throbbing violently"; but then he was seized, for the first time, by gout frankly in the foot, and on its appearance all the cardiac disturbance ceased. The pulse became regular, at a rate of 60, and the relief so far has proved permanent. Previous treatment addressed to gastric disorder had done some good. (Compare case, p. 503.)

This most interesting case suggested to me that the gouty poison which affected the heart so turbulently, not after the manner of angina, might have attacked the aorta after the transient fashion of gouty inflammations. I may mention here another case, to which I shall refer again under Diagnosis, in which an elderly man of almost unbroken health and activity had a slight attack of gout, which was followed by epigastric, and even slight thoracic oppressions, which seemed to differ from slight angina only in the cardiac arrhythmia associated with them. In him also after a few weeks' treatment the attacks wholly disappeared,

and he recovered his former bodily activity. If then it seems probable that gout, both as favouring arterial decay, and again by some more instant irritation, may take a considerable place in the causation of angina, we shall remember that authors on angina have for the most part written from experience of the wealthier classes in whom many other causes are at work. Diagnosis may be at fault. And we must not forget that even among the wealthy not a few anginous patients are of spare and temperate habit, with no signs or likelihood of gout.

If, as I shall argue, in the majority of cases angina pectoris be due to an "atheromatous" process of a senile kind, penetrating more deeply than usual in the suprasigmoid section of the aorta, a process scarcely to be called infectious in a definite use of the word, yet a considerable minority of cases, a number rapidly gaining recognition under modern clinical analysis, and entering into a class most significant in respect of the pathology of the disease, arise under the corrosive effects upon this part of *acuter and more specific infectious poisons*. As we have seen in the section on Aortitis, Flexner and other pathologists have found, besides the syphilitic spirochaeta, other specific microbes in aortitis, lying within the tissues, either unmixed or associated with septic streptococci. Boinet also has found in such conditions the microbes of erysipelas, of rheumatic fever (Poynton), and of influenza; in some cases grafted upon or turning into atheroma. Sir William Osler,[1] though he speaks sceptically on the clinical side, yet admits (p. 430) the frequency of infective foci of arterial degeneration, as in scarlet fever, measles, diphtheria, influenza, smallpox, and typhoid; foci recognisable on necropsy as patches in the aorta and larger arteries, or as sclerosis of smaller vessels. By one observer, quoted by him, such evidence was found in the bodies of 80 out of 300 children who had died of acute infections. But of all these infections the most active, definite, and frequent cause of angina is syphilis; after syphilis come influenza and rheumatic fever, and, if we allow for the irregular prevalence of influenza, in this order. But of these

[1] Article on Diseases of Arteries in Osler and Macrae, *Syst. Med.* vol. iv. c. 10.

infections I have written at length in the chapters Arteriosclerosis (Vol. I. p. 282) and Aortitis (p. 148).

The incidence of *syphilis* upon the aorta, and especially upon its suprasigmoid part is at a far higher rate than that of influenza, even during its epidemic periods. Burwinkel in his study (*loc. cit.*) of 117 cases, put the causes of angina in the following order: (1) syphilis; (2) obesity, diabetes, and gout; (3) aortic disease; (4) influenza and malaria; (5) heredity. Many cases of angina, apparently syphilitic, may be picked out of the records of the older writers. I must content myself with a few, such as this from Morgagni (*Ep.* iv. 22; see also *Ep.* xvi.), who emphasised lues as a cause of arterial disease:

Male, æt. 43. "Valde anxius," laid hand "on sternum." Died suddenly. P.M. Semilunar valve harder than normal. First portion of arch of aorta, as seen from outside, bulging *non secus ac si in tuberum modo quadamtenus hic illic assurgeret.* Inside, *superficies rugosa.*

We may compare this with Hodgson's, Welch's, Wilks', and later descriptions. His case of a woman who on the least effort suffered from pain in the upper part of the chest and left arm, who died suddenly in a paroxysm, and whose aorta was atheromatous, was probably syphilitic, as her age was only 42.[1] Lancisi [2] fully recognised the syphilitic or "Gallic" aneurysm; *e.g.* as follows:

Male, æt. 45. "Habitus carnosi, vitæ omnino libertinæ nimirum, Dianæ, Neptuno, ac Veneri frequenter indulgens, ex qua postrema pluris contraxit luis stigmata," etc. Again, an "egregius citharædus, qui ex impuro concubitu non semel in pudendis vulneratus." In both of these patients aneurysm followed.

In another paragraph, unfortunately on speculative grounds, or misled by the abuse of it in syphilis, he includes mercury as a cause of aneurysm, and deprecates its use; "in nullo Aneurysmate, etiamsi a lue gallico profecto." Senac quotes from Portal, "Cadaver syphilitici . . . aorta supra cor valde dilatata." Syphilitic aneurysm we all know, diffuse syphilitic disease of the arch we know, but syphilitic angina pectoris, depending

[1] See also Parkes Weber's case, pp. 187 and 264.
[2] Lancisi, *De motu cordis et aneurysmatibus*, Napoli, 1738.

upon syphilitic lesion of the supersigmoid and sigmoid area, has not received the general and prompt recognition which its frequency and urgent importance demands.[1] My friend of old days Lockhart Clarke [2] described an unmistakable case of syphilitic angina pectoris in which the aorta just above the aortic valve presented a raised convex transverse eminence with a somewhat rough surface (p. 187). It "consisted chiefly of fibrous tissue without any appearance of atheroma." The orifices of the coronary arteries were occluded. Sir William Osler (in *Lumleian Lect.*) said syphilis was not concerned in any one of his cases, which surprised me. May it not have been a factor in his case of the woman, æt. 37, cited later in the same lecture ? Dr. Mitchell Bruce [3] said that in his experience angina was nearly three times more frequent in the syphilitic than in any other degenerations of *the root of the aorta* (italics mine). From my own experience in England I must regard its recognition as of pressing importance ; for this kind of angina, if not promptly curable, is fairly amenable to specific remedies. Bouchard, at the Meeting of the British Medical Association in 1908, said that of 261 consecutive cases of syphilis 12 (more than 5 per cent) had angina pectoris ; while of 3739 consecutive cases of all kinds, its incidence was 71 (*i.e.* under 2 per cent) ; but of these 38 showed signs of aortic disease. In 7 of the syphilitic cases an aortic regurgitant murmur disappeared under active specific treatment (cf. case p. 179).[4] And not only is due vigilance called for in overt cases of angina which may have a syphilitic origin, but no less in slighter cases of stenocardia (angina minor), cases which may be so slight as to escape the serious attention of patient or physician, but yet may be no less significant of the same baleful poison, and no less perilous. As Mitchell Bruce says, a transient and equivocal

[1] The pioneers in this pathological research were of course Wilks (in 1863) and Welch. Hodgson may not have perceived that the gross disease of the aorta which he so well described was syphilitic. He scarcely says so. Among later tracts on the subject, are those of Heller, "Syph. Aortitis," *Verhandlungen d. deutsch. path. Gesellsch.*, 1900, Bd. ii. ; of Doehle, *Deutsche Arch. f. klin. Med.* Bd. lv. p. 190, a most important article ; and of Saathoff,—also of the Kiel School,—*Münch. med. Wochenschr.*, 1906, No. 42 (see p. 168 *et seq.*).

[2] Clarke, L., *St. George's Hosp. Rpts.*, 1866.

[3] Bruce, M., in his third *Lumleian Lecture.*

[4] See *Lancet*, Aug. 8, 1908 A more than usually lucky percentage.

sense of substernal constriction may to the wise be a hint of an initial stage of syphilitic aortitis which, in later stages, may, and too often does, proceed, if not to overt angina, yet to extensive deformation of the vessel, to impairment of its valve, or to the establishment of saccular aneurysm.[1] Unfortunately, when syphilis attacks the valvular area it is too often with the result of insufficiency ; syphilis rarely produces obstruction only, as atheroma does. Let us take a lesson from the following records of this accurate clinician. Of 28 cases from his own private notes of cardiovascular disease with a history of syphilis, Dr. Mitchell Bruce [2] found double aortic disease in 9 ; and practically all the rest presented that tone of the aortic second sound, which signifies degeneration in this area, while "apical murmurs were relatively uncommon." Two-thirds of them presented sclerosed radial arteries. "*Pain of anginal type was a prominent complaint in half the cases*" (italics mine). Was it in fear of the orthodox coronarians that the learned author did not allow himself the use of the plain name of angina pectoris ? He joined Dr. Mott in the warning that all this is imperfectly realised. In a case of syphilitic cardiovascular disease in a man, æt. 42, published by Sir Thomas Barlow,[3] attacks of angina pectoris set in, and persisted intermittently till death, two years later : at the necropsy was found syphilitic infiltration in the wall of the left ventricle, and "around the aortic orifice" ; there was also in this area a small unruptured aneurysm. It is remarkable that in this case the infection had occurred only three years before ; we shall see that the primary disease is usually farther back in time, even 20 years being no unusual interval ; though, as I have said before, we must not forget that in their confessions patients are tempted to push the sin, if such were its origin, as far back into the past as may be plausible. Three at least of my cases of typical angina of syphilitic nature occurred in physicians infected in

[1] See paragraphs on Angina and Aneurysm, p. 205, in the section in this work on Aortitis (my *Cavendish Lecture* of 1903) ; also Mitchell Bruce, *Lancet*, July 8, 1911, p. 74, an article in which he reinforces his lesson with added materials.

[2] *Lettsomian Lecture* for 1901 ; in substance repeated with more facts in *Lumleian Lecture* for 1911. (*Lancet*, July 15, 1911.)

[3] Barlow, T., *Trans. Pathol. Soc.*, 1877.

the course of their duty. One of them, whom Dr. J. Mackenzie will remember, was a lady. I recall at this moment only two cases in married women of syphilitic angina definitely due to marital infection. In Bruce's cases the average interval between infection and aortic symptoms was 25 years; with a maximum of 54 years and a minimum of 5 years. I have quoted his maxim that syphilitic aortitis may exist, and progress slowly and latently for years, "*as a rule, in the form of presternal oppression on exertion,*" before declaring itself (italics mine). The following remarkable and crucial case was very kindly communicated to me by Dr. Sidney Phillips:[1]

Male, æt. 34, syphilis in 1885. Never had rheumatism. Entered St. Mary's, October 31, 1895. Had had four attacks of angina pectoris, radiating especially into the throat. He had on these occasions "to crawl home." There was a diastolic murmur at the right base. On mercury and potassium iodide he made some improvement; but on December 23 he had a very violent seizure, and two days later died suddenly. P.M. Gumma projecting into the lumen of aorta immediately behind the valve; none of the cusps involved, nor independently diseased. There was about $\frac{1}{8}$ of an inch space between the margin of the growth and that of the cusps.

I will quote also the following case in a private letter from Dr. Rolleston:[2]

Male, æt. 27. In-patient, more than once, for angina pectoris (*supposed to be "functional"* !); last admission on October 8, 1890. Systolic apex murmur. The patient died suddenly after dinner on October 9. P.M. Ventricles dilated. Aortic orifice stenosed by extensive disease of the aorta (presumably syphilitic) immediately above the valve, which was thickened and could scarcely have closed properly. In this case, it is true, there was stenosis (see p. 263).

As it often happens, the orifices of the coronary arteries were in this case almost obliterated; these arteries being themselves otherwise normal. On the evening when this lecture was delivered in Fitzroy Square Dr. Parkes Weber had happily secured the heart and aorta from a fatal case of syphilitic angina (Female, æt. 42).[3] The specimen presented

[1] Letter dated Jan. 20, 1906.
[2] Letter to me, July 23, 1907.
[3] This case was published by Dr. Weber, *Proc. Roy. Soc. Med.*, April 1908.

the usual cushion-like protrusion of suprasigmoid luetic aortitis, and the process had advanced to occlusion of the coronary orifices (p. 182). I ventured to turn the case to my own purpose by paraphrasing Dr. Weber's interpretation; namely, that to the occlusion of the coronaries was due, not the patient's angina, but her death from angina; the source of the angina itself being the aortitis. Disease of any kind in the suprasigmoid part of the aorta is of course prone to invade the coronary orifices, and this condition it is which brings about, not the angina, but the mortal issue of it; yet many, or most, published records of angina are useless for my present research, because the pathologist, content with the discovery of coronary disease, or of gumma of the myocardium, gives little or no heed to the aorta. However in not a few crucial autopsies after angina we have clear evidence that the coronaries had escaped; as we shall see presently. But I am anticipating.

If our sight could penetrate the chest during life, we should recognise cases of angina without coronary implication more frequently; for, as most of the syphilitic and other infectious cases are in comparatively young persons, the disease in them is often not mortal; at any rate not by anginal inhibition, though only too often death ensues indirectly by way of aneurysm or of aortic regurgitation. Here however is a case in a male, æt. 60, a patient of Dr. Mathewman of Bromley:

Infection "25-30 years ago." For five years (in the secondary stage) persistent skilful treatment. Accessible arteries normal. Systolic murmur at base, and altered second sound. Systolic pressure 140. *Has had "pulsus alternans" for a long time; showed us a tracing of it by Dr. Newton Pitt taken 14 months before.* This persists. No dilatation of heart. No Stokes-Adams symptoms. Angina pectoris in typical form, but not very severe in degree, and abating.

In syphilitic, as in other infectious cases, pressures do not as a rule run high; the patients are for the most part under middle age. But in the following patient, æt. 50, sent by Dr. Eve of Hull, Hyperpiesia and Angina were in co-operation.

He had lived generously, plenty of wine, etc. No tobacco. Some years ago specific infection, non-suppurating bubo. Wife had

miscarriages. Had careful antisyphilitic treatment. Two years ago, walking after partridges, pain in left forearm; it continued as he walked for an hour or more, but did not pull him up. Soon however the pain did begin to arrest him, even on the slightest ascent; and it extended up inner arm to shoulder, and crossed the sternum about the manubrial joint. Did not mention dread. Arteries rather thick. Systolic blood pressures about 200. (Eve and myself, 190 and 200.) No definite signs of renal disease, though a trace of albumin had been detected occasionally. No casts. First cardiac sound with faint murmur at base. Aortic dulness, at manubrium and to right of it, very definite. The general treatment (besides the specific) had been rigid dietary and laxatives (Eve).

It is therefore of the utmost practical importance, in examining a case of angina pectoris, especially in a patient not obviously a subject of senile atheroma, to search for any note of old syphilis; a characteristic scar, however tiny, or mark on limb, eye, or pharynx; pupils insensible to light; a premature general arteriosclerosis, a reaction to Wassermann's test, and so forth. In all cases of angina under the age of 60, in which evidence of syphilis is not patent, the Wassermann test should be applied.[1] I have said that syphilitic aortitis is in my experience not always accompanied by a general arteriosclerosis; Dr. Mitchell Bruce found this in one-third only of his cases of cardiac syphilis; he puts it "that syphilis of the organs of the circulation is a locally distributed disease." Dr. Mott's experience is to the same effect.[2] Even if, with the angina, nephritis high-blood pressure and arteriosclerosis seem to complete a diagnosis of Bright's disease, we must not forget that recent researches [3] have suggested that nephritis is one of the consequences of syphilis, so that this subtle infection may still lie at the bottom of the case, and be a proper object of the treatment. Again and again it has forced itself upon my mind that symptoms interpreted as thoracic or cardiac crises of tabes, were sometimes those of a coincident angina pectoris engendered by the same poison; a distinction not to be regarded as a mere clinical refinement, because a vigorous specific treatment, if bootless in respect of the tabes, may not only dissipate the angina, but also prevent

[1] See Jacobaeus, *Deutsche Arch. f. klin. Med.* Bd. cii., 1911.
[2] My own experience, essay on Arteriosclerosis (Vol. I. p. 295, etc.
[3] Sir John Rose Bradford, and others.

extension of the mischief to the aortic valve (p. 176). How latent the aortic disease may be is illustrated by this case of Dr. Morison's,[1] which I give in summary:

Male, æt. 32, Angina ("typical") one month. Physical signs negative. No abnormal sign about the base of the heart. Urine normal. Died in an intense and protracted seizure. P.M. Typical Hodgson's aorta, which had "nearly occluded the orifice of *one* coronary artery." *Myocardium* "*perfectly healthy*" (italics mine). "Aortic valve normal; cardiac plexus normal" (*vide* p. 362).

Among many other cases of syphilitic angina on record, I wish to refer to two, very similar, published by the late Professor Dieulafoy;[2] for the one which ended in recovery was verified, if I may say so, by the second which was mortal; in this instance again after death suprasigmoid aortitis only was found. Dieulafoy was compelled to admit that disease in this spot can set up angina pectoris without any implication of the coronaries.

In the number of cases, no inconsiderable number, in which life was not forfeited, we may guess that the disease did not invade the aortic cusps, nor block the coronary arteries; or that specific treatment was adopted in such good time that any such exudation was dissolved; or again that these vessels, if blocked wholly or partially at their orifices, were still permeable, or could accept blood from other connections. If in cases with aortic regurgitation sudden death occurs, it may be impossible to say whether death were due to the aortic or to the coronary disease, or to both. I have already insisted (p. 21) that extreme stenosis or occlusion of the coronary orifices is not inconsistent with an apparently normal myocardium; though, if with closed coronary orifices life and a fair integrity of the heart are to be spared, the process, as we have seen in the chapter on Cardiosclerosis (p. 23), must be very slow. It seems at first sight that syphilitic aortitis would go too quickly for such accommodation, yet I have seen some recoveries from severe syphilitic aortitis, in which specific treatment had not been very prompt, or not very systematic. The misfortune is that too often a permanent aortic regurgitation is established (p. 182); usually,

[1] Morison, A., *Cardiac Pain*, p. 35.
[2] Dieulafoy, *Traité*, etc., 15th ed. pp. 983-984.

says Tripier,[1] with separation of the cusps from each other at the points of attachment (p. 186). This I also have observed.

Even after recovery from syphilitic angina, we must warn the patient, as in other forms of syphilis, against a relapse of the malady. This misfortune was painfully impressed upon me in the case of a gentleman who, at about æt. 45, was under my care in 1906 for more than a year, during which period we got rid of the angina, and of all signs of active syphilis. But in 1910 a recrudescence of the angina set in with a faint aortic regurgitant murmur. The patient then came under the care of Dr. Evans of Lowestoft, with whom I corresponded. In spite of energetic specific treatment he died, and with suffocative symptoms which Dr. Evans attributed either to latent aneurysmal pressure on one bronchus, or to mediastinal syphilis.

I have suggested that in some of these cases the swelling might not have invaded the mouths of the coronary arteries, or had receded from them. As, during a comparatively acute aortitis, the process is extending to the valve, the second sound, as we have seen (p. 200), is apt to alter in a peculiar way ("bruit de tabourka"), different from that of atherous conditions. When the valve is touched, incompetency, with a double aortic murmur, soon appears; an order which Dr. Mitchell Bruce is right in contrasting with that of ordinary—"gouty or senile"—atheroma, in which the murmur is usually systolic only.

Tuberculosis has not much to do with angina pectoris; but here I must cite a remarkable case of an old phthisical patient quoted by Dr. Ewart from Perez, a case of mediastino-pericarditis in which "Perez' sign" was heard. The case has been commented upon by Ewart,[2] Powell, Fowler, and others. The patient suffered from "excruciating, in fact anginal, pain." There was "no reasonable doubt that inflammatory adhesions of a chronic tuberculous nature were present in the anterior mediastinum." (For Malarious Aortitis, see p. 165.)

Influenza.—The severe epidemic at the end of the nineteenth century brought more into light the occasional attacks of this infection upon the aorta; and if, fortunately, in proportion to the diffusion of the disease itself, and even perhaps to some

[1] Tripier, *Études anatomo-cliniques*, Paris, 1909.
[2] Ewart, W., *Brit. Med. Journ.*, April 6, 1912.

latent degrees of influenzal aortitis, angina pectoris is rare, yet the absolute number of recorded cases of this dangerous sequel is far from inconsiderable. This sequel I have discussed in the chapter on Aortitis (p. 163), and will here touch only upon its connections with angina.

Influenza was regarded by Gibson as the chief of the infectious causes of angina; but, in this infection, as in rheumatism, the attacks of angina are often attributed, as apparently by Gibson himself,[1] to a myocarditis with painful stretching of a weakened ventricle. Yet in diphtheria, although myocardial softening and dilatation are recognised phases of the disease, angina, if not unknown,[2] is very rare. The attacks, Gibson adds, *resemble in the closest manner true angina* (italics mine); . . . though he "could not but conclude that the cause of these symptoms was some nervous disorder, and not a true angina pectoris." And, as I have said elsewhere, Sir William Osler's and Dr. Shadwell's records of "spurious angina" in influenza, justifiable as their diagnoses may seem, do not exclude the genuine disease as no very rare sequel. Sansom's papers on this subject,[3] of which I made free use in my *Cavendish Lecture*, were at the time of their publication the most important exposition of this interesting subject, and one the more impartial, as Sansom was at first beset by the reigning opinion that the attacks, typical as they were, could not be angina pectoris because his first cases were not mortal, and because the heart seemed "perfectly normal." Then however came two mortal cases, and in them patches of subacute suprasigmoid aortitis were revealed; so that in his subsequent experience of such cases, whether mortal or not, Sansom accepted with more confidence the aortic origin of the angina. He described the morbid histology of three cases of aortitis, but they presented little to distinguish them from acute aortitis arising from other causes. On March 7, 1908, Dr. Finney of Dublin wrote to me to ask the question, "Have you met with fatal angina after influenza, with a weak dilated left ventricle? One of my patients died in this condition, and another is now (in peril). . . . There

[1] Gibson, G. A., *Disease of the Heart and Aorta*, p. 764.

[2] See Aortitis, p. 164.

[3] Sansom, E., *Trans. Med. Chirurg. Society*, 1894 (following a paper two years before); and *Hunterian Lecture* of 1899 on "The Effects of Influenza upon the Heart and Circulation" (*Lancet*, Oct. 21, 1899).

was no valvular lesion in either case." Merklen again testifies that angina pectoris follows "la grippe . . . assez souvent." [1] Marmorstein, in a remarkable article, "Des aortites grippales," [2] gives, as a chief symptom of this aortitis, "douleurs avec caractère angoissante et rétrosternale." Furthermore, we are now aware that not myocarditis only but endocarditis and pericarditis also may arise in this disease.[3] Sir William Osler, who in his textbook [4] says "a number of cases of angina pectoris have followed influenza," in another place [5] narrates the case of Chief-Justice X., who in the winter of 1893 had a severe attack of influenza. During convalescence he felt pain in the chest on ascending hills—attacks of angina, which became more importunate and even severe. With some intervals of relief in the summer season, the angina hung about him, and in the winter gained upon him. *He died in an attack.* The patient, says the author, always insisted that the influenza was the cause of the angina. In the same place the author records another case, the sequel of influenza, which ended in recovery. It observed the usual areas of radiation, and the attacks were so horrible that the patient feared a respite lest he should have to go through the suffering again! That, notwithstanding phenomena so unequivocal, my distinguished colleague should have placed this case in the category of "pseudo-angina" is to me incomprehensible.

Curtice and Watson of Philadelphia [6] have discussed "The Action of the Influenza Poison on the Heart," and collected about 70 cases of influenzal angina pectoris. Some of these are of rather doubtful interpretation, but they confirm the maxim which I had proposed before, that when influenzal or other angina occurs in young and vigorous patients the large majority of them recover. They found that influenzal angina might be as severe as that due to any other cause; in some there is the peculiar dread, especially in the elderly cases, which indeed were often mortal. They give some interesting records of their own; one in a youth æt. 18. An important case, narrated by Rendu, will be found in the chapter on Diagnosis (p. 498). Dr. Guthrie

[1] Merklen, P., "Endo-arteritis bei Influenza," reference mislaid.
[2] Marmorstein, "Des aortites grippales," *Rev. de méd.*, 10 mars 1908.
[3] *E.g.* Flexner, S., *Penn. M. Bull.* xv. 1902-3. [4] Edition of 1905, p. 893.
[5] Osler, W., *Angina Pectoris*, p. 29.
[6] Curtice and Watson, *Internat. Med. Mag.*, January 1893.

Rankin [1] has reported a case, in a lady, æt. 48. During convalescence she felt for the first time "discomfort referable to her heart, and went through paroxysmal attacks of præcordial pain . . . suggestive of angina pectoris. The *aorta was beating in the episternal notch, and its throbbings were unpleasantly manifest*" (italics mine). She had a great dread of these anginal attacks, which, however, were relieved by vasodilators. There was no suspicion of syphilis. As the lady recovered, we cannot on the data before us be sure of the genuineness of this case, especially as the phrase "præcordial pain" is now used so equivocally; but at any rate the illness seems to have been very grave. Dr. Rankin likewise seems to have based a diagnosis of "pseudo-angina" on the fact of recovery; but I must reiterate that in persons whose hearts are sound, recovery from infective angina pectoris is the rule, if a rule with too many exceptions. A sad instance of such an exception occurred in the case of a Kentish physician, æt. 40, of sound constitution, who after influenza was attacked by angina pectoris. While at work in his surgery, so violent an attack seized him that he died in less than an hour. Yet if he had crawled back into life, and ultimately survived, no protestations would have saved him from the imputation of a "mere neurosis." Fränkel, in the fourth edition of *Eulenberg*, reports a fatal case of influenzal angina in a man æt. 50, who had presented no previous signs of cardiovascular defect. No autopsy is mentioned. Dr. Samuel West [2] says that "attacks of cardiac failure, which we now recognise as a rare sequel to influenza, were in earlier epidemics more frequent"; and he refers to cases in his own experience of "sudden feeble irregular heart, and imminent peril of death, or actual death"; symptoms, by the way, which, as we have seen, are not those of angina—unless any cardiac dissolution may be thrown into angina,—but with these he describes also some cases "of anginal form, *often having the same character as true angina* (italics mine), and radiating in a similar way, or with increasing feelings of tightness or oppression, associated with a sense of cardiac syncope." Here again I declare that if the cases have the same characters and issues as angina, characters in which a physician of Dr. West's

[1] Rankin, Guthrie, *Practitioner*, May 1904.
[2] West, S., *Practitioner*, Jan. 1907 (Influenza Number).

experience and ability can read no difference, the refusal to admit them into the category of angina becomes, in a Euclidean sense, absurd.

As in the more familiar modes of angina, the vasomotor symptoms which may accompany it are secondary. For instance, during an attack of influenzal angina, in a case in which aortitis was verified after death, Sansom noted that "the radial artery contracted, and the mucous membranes became pallid; the vessels then dilated, and the face and lips flushed"; tides which may attend upon the irritation of any afferent nerves. In the behaviour of this or that case fallacy may lie; but when again and again these cases are agonising, perilous, and even mortal; when, concerning such a case, a medical colleague in his letter to me says that it is one of "pseudo-angina," although, he adds, "the attacks were extremely like those of true angina, and are induced by slight exertion"; and when in not a few necropsies an underlying acute aortitis has been demonstrated,[1] and yet, notwithstanding these agonies and these perils and these findings, an accomplished physician of the day writes, of the sequels of influenza, that "*various cardiac neuroses* (italics mine), such as bradycardia, tachycardia, and angina (!) are only too commonly the result of this disease," and when his disciples take upon themselves the grave responsibility of assuring such patients, and the friends of such patients, that the symptoms are those only of a mock angina pectoris—a mere nervous storm—does not equivocation become worse than "absurd"?

On the contrary, Sir Richard Douglas Powell,[2] whose opinion on these subjects carries great weight, testifies that he has seen "toward the decline of an attack of influenza, acute anginal seizures; no cardiac lesion, yet severe anginal cramps of the heart." Of my own experience let me recall the striking case which I saw with Dr. Humphry (Aortitis, p. 163): and to it I will add the following brief summary of a case which, during the last influenza epidemic, I saw with Messrs. Hartley and Agnew of Bishop Stortford:

Mr. T., æt. 63; attack of influenza with pyrexia for a few days—highest recorded temperature about 102°. The fever had not long

[1] See *Cavendish Lecture*, p. 163.
[2] Powell, Sir R. D., *Practitioner, loc. cit.*

fallen, when one evening about 10 P.M. he had a violent attack of angina, and a slight systolic aortic murmur appeared. I saw him on the fourth day thereafter. The aortic murmur was now quite loud, and the angina pectoris had passed into the status anginosus; though the agony had been palliated by nitrites, morphia injections, and absolute rest. By such expedients for ten days the malady was scotched, and after this time gradually disappeared. He never had any dyspnea, and the pulse, save rises of rate now and then under pain to 88-90, was normal throughout. The pain was substernal, and radiated down the left arm, especially to one spot on the inner aspect of the upper arm a little above the elbow.

Now three weeks before this influenza, Mr. T. had been carefully examined for a large life insurance, and accepted at ordinary rates. Had the systolic murmur been there, it could not have escaped observation, as it was loud over a large area. I noted also a definite extension of dulness to percussion on the manubrium and two fingers' breadth to the right of it.

Cooper [1] has described a case of perforating aortitis in which "there was no evidence of syphilis or injury, but a probable history of influenza shortly before the aortic symptoms. The aorta was adherent to the œsophagus, left lung, and pleura. In the aorta was a round well-defined hole about an inch in diameter. It appeared to be "a circumscribed aortitis."

In *influenza arteritis* may likewise befall *other vessels.* Edmond Fort [2] records 6 deaths in 15 cases of this kind. The arteritis is not dissimilar from that of other infectious diseases. It frequently occurs during convalescence, and is most frequent in the arteries of the leg, generally on one side; but it may be bilateral. Gangrene may ensue. The pain may be so severe as to demand the injection of morphine.

Of *malaria* as a cause of angina, I know nothing. (But see Aortitis, p. 165.) It is confidently alleged as a cause by some authors, as by Le Roy de Méricourt and Laveran. However, men on travel, or foreign service, are apt to pick up other infections beside malaria.

From a recent article (I think by a Russian physician, but

[1] Cooper, R., *Lancet*, Feb. 21, 1914.

[2] Fort, E., Thèse de Paris, 1901. Summary in *Epit. Brit. Med. Journal* of that year.

I have lost the reference) I learn that in convalescence from *plague* anginal seizures may appear (Aortitis ?).

Rheumatic aortitis is more frequent than is generally supposed. Being usually superficial it is often painless. In the pages of careful clinical observers we may note here and there, as, *e.g.* of Merklen,[1] " violente douleur retrosternale avec oppression " ; indeed he accepts angina as a rare event in aortic rheumatism ; but his attention is so riveted on the heart that the significance of the sign is unheeded. However, as on this event and its connection with angina pectoris I have written already at sufficient length in the section on Aortitis, I must be content here with little more than a reference to it (p. 150). But I may repeat that Dr. Carey Coombs,[2] in his researches into rheumatic myocarditis, states that " *aortitis is usually present* " (italics mine) at the root of the aorta which " microscopically proves to be inflammatory " (p. 159). It is proper to add that he detected also arteritis of the coronary arteries. I have mentioned Dr. Hugh Stewart's[3] report of a case of angina pectoris in a boy, aged 7, suffering from severe rheumatic aortitis ; the pain came on suddenly with pallor and anxiety. Also (p. 158) a case of the same combination in an undergraduate under the care of Dr. Christian Simpson, one of the most excruciating cases (for the time) of angina I have ever witnessed ; and I have seen since another case, not so severe but typical enough, in a girl aged $12\frac{1}{2}$. Dr. Simpson's patient made a complete recovery, save for an aortic regurgitant murmur. He was ordained, and undertook full work as a curate near London. About ten years later he died suddenly. And I would again remind the reader of the very interesting case of angina minor in a young man suffering from acute rheumatism, under the care of Dr. Windsor of Royston, to which I have referred in the section on Aortitis (p. 157).

Dr. Poynton[4] narrates two very interesting cases of " acute inflammation of the aorta, associated with profound myocardial disease." I must permit myself to transcribe the introductory paragraph :

[1] Merklen, *Leçons*, 1908, p. 172, as quotation from " Freund " ; no more accurate reference given.

[2] Coombs, Carey, *loc. cit.* p. 159 ; and *Quart. Journ. Med.*

[3] Stewart, Hugh, *loc. cit.* p. 154.

[4] Poynton, *Lancet*, May 20, 1899.

"Cases are recorded from time to time in which, with or without angina, death occurs quite suddenly, and in which the necropsy reveals a condition of inflammation at the base of the aorta, unusually acute in its character and remarkably localised in its distribution. In these cases the aortic valves may be thickened by chronic inflammation, and yet they may nevertheless be quite competent, and neither during life nor after death can aortic regurgitation be demonstrated and brought forward as an explanation of the sudden cardiac failure. The area of the aorta which is so gravely and severely diseased is the one from which the coronary vessels arise, and it is a natural suggestion that the heart-wall has suffered in its nutrition either from a narrowing of the lumen of one or both of these arteries, or that the vessels have shared in the same acute process, and their ramifications have become occluded as a result. A reference to the literature upon this subject proves that such may be the case in some instances, but in the two cases which are recorded below I do not think that such an explanation is tenable. In neither of these two cases were the orifices of the coronary vessels certainly diminished in size, nor were their walls visibly diseased; and microscopic examination of the heart-wall showed that their ramifications were not occluded. In both cases the cardiac wall was examined with especial care, and in both the changes found were widespread and severe, and of a nature analogous to that found in the aorta. It is difficult to escape from the conviction that there had been a common antecedent cause which had thus injured these two organs which anatomically and physiologically are so closely associated. The changes in the aorta were obtrusive, but in the first of the recorded cases those in the heart-wall might macroscopically have been easily overlooked; yet in both cases I should suppose the sudden death depended far more upon these less obtrusive signs in the heart than they did upon the striking changes in the vessel-wall. The question of the etiology of this condition is one of great difficulty, and it will perhaps be the most rational course to record the two cases with the necropsies and microscopy before allusion is made to their causation."

Of the first of these cases Dr. Poynton gives the full clinical notes; from these I will quote but this sentence: "Dr. Cheadle saw her during this attack, and considered it to be one of true angina." After death the aorta in its first two inches appeared to be the seat of an acute, supervening upon a more chronic, inflammation. The branches were not affected. *The orifices of the coronary arteries were open*, the walls of both vessels were macroscopically healthy, and (in a number of sections) no occlu-

sions were detected. No micro-organisms were detected in heart or aorta. Of the second case no clinical history was obtained. The death in both was ascribed by Dr. Poynton wholly to the lesion of the myocardium, in my opinion an incomplete conclusion. Later, Poynton and Paine [1] say of these cases: "The end is usually quite sudden, and the necropsy shows as its most striking feature an acute inflammation of the aorta." Among my own notes I find the following case (here much abbreviated):

Miss H., æt. 47. At the age of 18 had rheumatic fever, "and with it, and for a short time afterwards, had angina pectoris." This information she gave me unasked, and said the diagnosis was made without hesitation by two eminent physicians. (The neurotic notions were not then in vogue.) The angina had never returned, but heart symptoms had been coming on of late. She has now a dilated heart and a mitral murmur with the usual signs of œdema, etc.

It is, perhaps, a matter of surprise that angina pectoris does not more frequently accompany *malignant endocarditis*, a process which is often engrafted upon the aorta from its valve, or arises there independently. Still there are not a few such cases on record, and two at least have fallen under my own eye. The case quoted from Dr. Morison (p. 301) as of dextral radiation was one of malignant endocarditis.[2] A good description of this incidence is given in Charcot and Bouchard's *System of Medicine* (ed. 1893) by Dr. André Petit. He says an ulcerative aortitis, consisting in an extension of the infectious process to the aorta from the endocardium, presents symptoms coincident with those of the endocarditis; [3] but sometimes manifests itself by crises of aortic pain of a *pseudo-anginous* (!) character, radiating into the back and shoulders, sometimes *by seizures of true angina pectoris* ("par accès de véritable angor pectoris"), a fanciful distinction; indeed he adds, "In some cases an appreciable dilatation of the first portion of this vessel can be demonstrated." Passing over the inconsistency of this writer's inferences, I can testify to the truth of his description; and that after death the inflammation may be verified as a propagation of the infectious process from

[1] Poynton and Paine, *Researches on Rheumatism*, 8vo, 1913, p. 67.
[2] From information kindly sent to me by Dr. Morison in a private letter.
[3] *Vide* Aortitis, p. 161.

the valves and orifice to the tube of the aorta. In such cases the coronary orifices may not be involved. As regards the inconsistency of diagnosis, even Dr. Stengel surely entangles himself in a like distraction of meaning when, as if using the very phrase of Petit,[1] he says that aortitis gives rise to "spurious angina"; yet describes the pain as substernal, radiating to the shoulder, etc. ". . . *without the involvement of the heart muscle*, so often seen in true angina." "These patients," he adds, "are especially liable to painful attacks on any physical exertion." Just so; then where during life is the criterion of spuriousness? Of what use to the traveller is a signpost invisible while on his journey; or to clinical medicine a diagnosis which cannot be made till the patient is dead! It is not quite clear indeed what that is in which the heart muscle in "true angina" is found "involved"; my friend Dr. Stengel cannot mean that in this malady the disease of the heart muscle is a pathological criterion or correlation of true angina, for, if so, why "often"? Either myocardial disease is essential to angina or it is not; Dr. Stengel's words imply that it is not, which is true. In death from angina pectoris the state of the heart, whether normal or abnormal, is not a constant.

Surely all this pseudonymous hair-splitting does not tend to edification, for in rheumatic, influenzal, and other infectious forms of aortitis the angina is generally attributed not to a false but to an organic lesion; namely, to a myocarditis accompanying the aortic disease. Sir J. W. Moore,[2] for instance, and Fräntzel[3] likewise, have attributed attacks of angina pectoris to the weakness and dilatation of the heart, consequent upon the acute infection. Is it not sufficient, on the other hand, to point out that in certain infections, such as diphtheria and typhoid, which seem very rarely to touch the aorta, but do commonly poison the heart's muscle, angina occurs not "often," but indeed very rarely, and from the cardiac disease never?

Dr. Parkes Weber asked me why angina was not an invariable effect of, say, syphilitic aortitis. Well, as we shall see, much depends on the rate, the intensity, and the precise seat of the

[1] *Loc. cit.* p. 31.
[2] Moore, Sir J. W., *Dublin Med. Journal*, 1890.
[3] Fräntzel, *Vorles. d. Krank. d. Herzens*, 1889.

lesion. The aortitis may be too superficial; or the afferent endings may be destroyed so gradually that pain may be averted or slighted, or some readjustment brought about. I may ask in my turn why angina does not occur in all (or in most) cases where the coronary arteries are occluded? This inconsistency Dr. Weber admits in the *Pathological Transactions*, 1906, where he gives an explanation he did not offer to me.

CHAPTER V

SYMPTOMS

As in the introductory sections of this essay I have divested angina pectoris, or shown how to divest it, of the false draperies which of late have been the fashion, now let us attempt to delineate its features in their true austerity. Angina pectoris has been too often and too admirably described to need any new portrayal:[1] I propose first to discuss its symptoms so far only as may be necessary to illustrate my theme; next, to make a similar selection from its morbid anatomy; then, upon the facts thus reconsidered, to erect my own hypothesis, and, finally, to deduce therefrom what may be relevant concerning diagnosis and treatment. Although we decline to recognise phantom anginas, yet angina pectoris itself, often in typical, often in larval or fractional, forms, presents to us, as do all other diseases, symptoms and by-symptoms, main symptoms and fringe symptoms, symptoms essential and symptoms incidental. Let us begin by distinguishing the essential symptoms, those which are cardinal and crucial, and consider the subordinate symptoms afterwards. In the first place, then:—

A. Cardinal and crucial symptoms are:

(i.) Peculiar and startling pain, usually sternal in seat but, as it forces its path into contiguous segments

[1] I have skimmed through Hippocrates, Celsus, Aretaeus, parts of Galen, Rufus, Oribasius, etc., and failed to discover any definite record of the disease; therefore I have omitted a historical section from this essay. Seneca's well-known case however seems unquestionably genuine (p. 319). The name "morbus cardiacus" covered a heterogeneous lot of cases; probably angina pectoris was among them. Here and there one comes across remedies for pain in the chest evidently very severe and alarming. Scribonius Largus speaks of this pain and of a secret remedy which he could not obtain till the death of him who held it; then it was published and dedicated to Tiberius.

of the spinal centres, above and below the last cervical and upper thoracic circuit, spreading thence into widening areas — into the left arm, or right arm, or both arms, the neck, and parts even still more distant, and frequently attended, or alternated, with paræsthetic sensations; sometimes traversing the segmental centres from sensory to motor tracts, to set up paresis or cramp of corresponding muscles, or even an atrophy of them.

(ii.) Arrest or restraint, brief but imperious, of respiration; and more or less of all muscular activity.

(iii.) Sense of impending death, not constant but in many cases appalling; often, even in slight or incipient cases, an organic dread or sense of ill-omen, as contrasted with rational apprehension.

B. Accidental or subsidiary features, not crucial nor cardinal:—

(i.) High arterial pressure; sometimes primary, sometimes secondary, often altogether absent, even from typical cases.

(ii.) Atheroma, or other general arterial disease.

(iii.) Vasomotor disorders, such as are seen in other painful or alarming conditions; polyuria, sweats, salivation, etc.

(iv.) Restlessness, belchings, tremor, dyspnea (in complicated cases).

(v.) Some discomposure, be it more or less, of the heart's rhythms or energy.

(vi.) Sudden death, by cardiac inhibition.

C. Aberrant or larval cases; in which, for example, the pain may for a while remain peripheral, as in a finger, or in one or both hands; or, very rarely, without pain, as a sense of sudden arrest and dread; or, again, in which the pain is felt in the epigastrium, or about the apex of the heart, or below it in the course of the seventh, eighth, or ninth thoracic nerve, or even still lower in the abdomen.

Pain. — By common consent we shall begin with the pain; and this in angina is not mere weight, oppression, or aching about the heart. The recent laxity in defining the seat of the pain counts for much in the prevalence of

erroneous opinion concerning the nature of angina pectoris; or rather the common fault is not so much a lack of precision as a certain precision of error. In one of the examinations for the Membership of the College of Physicians, a test of candidates of comparatively ripe clinical experience, in answer to a question—not of my setting—on angina pectoris, I found in every answer the pain described as "præcordial." Gibson said the pain "is often confined within the precordia"; though he himself had shown that it would be truer to say that it never is. In only one of his own diagrams did this area even partake of the pain, and this was in a complicated case with double aortic murmurs and a dilated heart.[1] Now strictly the "præcordia" are the diaphragm and parts about it; and its metonymic significations have been less in the direction of the heart than of the upper abdominal viscera—the liver, spleen, stomach. "Leni praecordia mulso dilueris melius." Thus we read severally of "pectus, cor, et praecordia."[2] By "angustia praecordiorum" the older writers meant, not angina—which may have been hinted at as an "insultus ferox," etc.—but the submammary depressing ache of chronic heart-disease. However, what our text-books and their readers intend by the word, as indeed they are wont alternatively to say, is "the submammary cardiac region," "at the heart," etc. Romberg uses the words "Herzgegend" and "Herzschmerz," plainly meaning the submammary area; Eichhorst says the chief symptom (Hauptsymptom) of angina pectoris is "Herzschmerz"; and Dr. Byrom Bramwell, no less an authority, defines angina pectoris as "A paroxysm characterised by pain in the region of the heart."[3] In a well-known modern English treatise the pain is described in like manner, as seated "at the heart," the words "in regione cordis" being added later, apparently as a quotation; and, a few lines

[1] Gibson, G. A., Fig. 9, p. 20 (*Edin. Med. Journ. loc. cit.*), and *Diseases of Heart.*

[2] Celsus uses "praecordia" rarely of the thoracic, generally of the abdominal cavity; for Pliny the word means viscera ("exta"). When Persius said of Horace "circum praecordia ludit," he was not referring to the submammary or any unilateral spot. If we agree to transfer the meaning of "praecordia" to the area of the heart, we must be quite clear as to our use. Or we might introduce the word "pericordia," which, in this sense, occurs here and there in ancient medical writings.

[3] Bramwell, Byrom, *Brit. Med. Journ.*, Jan. 15, 1910.

farther on, the author explains, with no notion of inconsistency, that the sternum is squeezed or crushed back to the spine as if with a vice. A medical friend of mine, in introducing to me a patient suffering from angina, said "his præcordial pain is terrible"; whereupon, by way of justifying his physician, the patient rubbed and patted the upper part of his sternum. A physician, writing from a foreign watering-place in high repute, refers the pain in a certain case of angina to "the region of the heart," yet in the very next sentence speaks of it as seated in the "upper third of the sternum, and the second and third intercostal spaces, radiating to the left arm and back." The better early authors, on the other hand, are consistent in speaking of the sternal, or substernal seat of the pain; the phrase "heart pain" is a modern blunder derived, not from the observation of nature, but from preconception; for it is remarkable that in all the vagaries of its distribution its rarest flight is to the submammary area. Now, by the tyranny of words, this false turn of phrase, biassed no doubt by the only too real sense of peril at the heart, has contributed more than we suppose to the stiff-necked assumption—for a sheer assumption it is—that the seat of angina pectoris—of the pain, that is, not of the death—is in the heart, and to the laboured ingenuity with which this interpretation is dressed up. A cardiac angina there may be, but we shall see that to distinguish it needs far more discrimination than is usually devoted to the problem.

For the clinical facts let us first consult Heberden: of the pain this leading authority writes:[1] "Sedes ejus est modo suprema pars, modo media, modo ima ossis sterni; non raro tamen inclinatior ad sinistrum quam ad dextrum latus." Of the sudden crushing and lancinating pain he says, "Pectus quasi constringente, qui sub sterno modo superiore, modo inferiore, in plurimis orsus est"; thence radiating to shoulder, arm, elbow, or hand. In Percival's case[2] the pain was "about the middle of the sternum, inclining to the left side"; and the same is noted in Alexander's case (both in the third volume of the *Edinburgh Medical Commentaries*). Heberden says emphatically that it is not easy to guess why the pain makes itself felt in these several

[1] Heberden, *Commentaries*, edit. 1807, p. 310.
[2] Percival, *Med. Observations and Inquiries*, vol. v. p. 236 (1773).

parts of the "os sterni," but such is his conclusion from "not fewer than a hundred patients suffering from this disease." Fothergill,[1] whose experience was considerable, is quite definite about the pain, a constrictive pain seated in the sternum, usually in the upper part, and passing across the left breast into the arm. Before Heberden, Morgagni had said of such a case (*Ep.* 23): "She was subject to attacks of violent anguish in the *upper part of the chest* on the left side, with numbness of the left arm"; and another (*Ep.* 4), "a certain divine used to point to his sternum as the seat of his pain, *a symptom which proceeded from a disease in the aorta, which lies deep in the breast under this bone*" (italics mine); Parry[2] describes the pain as mid-sternal, crossing to the left side; Jurine, as "douleurs sternales" . . . "à travers la poitrine"; Wall,[3] as a pain in the upper sternum, crossing one or both pectoral muscles; Wichmann,[4] as "a bar across the upper chest, the pain being as it were within the chest." So general is this feature that Baumès[5] of Montpellier, in 1808, proposed for the disease the name of "sternalgia." We note that none of these classical physicians uses the parrot-like and inaccurate word "præcordial"; they speak definitely of the pain as sternal or substernal. Trousseau, although in his general remarks on the disease he more than once speaks carelessly of "præcordial" pain, yet in the description of each of his cases severally, reports the pain as "à la base de la poitrine, derrière le sternum," adding categorically, "*Sans douleur sternale point d'angine de poitrine . . . si le cœur était affecté la sensation au début de paroxysmes était précordiale*" (italics mine). And the modern writers to whom we look for precision are no less careful. Broadbent, who did not copy other people, wrote: "the pain is chiefly behind the sternum, with radiations"; it is "vice-like; a tearing or burning" . . . a "gigantic weight" . . . "*usually* (italics mine) with a sense of

[1] Fothergill, edition of his works, 1783, and previously in *Med. Observations and Enquiries*, vol. v. (1773).

[2] Parry, C. H., *An Enquiry into the Symptoms and Causes of Syncope Anginosa, commonly called Angina Pectoris*, Bath, 1799.

[3] Wall, of Worcester, letter to Heberden, *Med. Trans.* vol. iii. p. 19, 1772.

[4] Wichmann, *Ideen zur Diagnostik*, Hanover, 1797, an excellent commentary on angina.

[5] Baumès, *Nosologie*, tome ii.; previously *Annales de la Soc. Méd. de Montpellier*, Oct. and Nov. 1808.

impending death." Sir William Osler, whose mind is trained on these earlier authors, speaks of "agonizing substernal radial pain," and *distinguishes it from the submammary ache of ordinary heart-stress.* In another passage he says, "the maximum intensity of the pain is substernal . . . in some at the xyphoid." Throughout his *Lumleian Lectures* he avoids the loose phrases of "præcordial pain," "pain in the region of the heart," etc.; and affirms that heart pain has not the intensity, the paroxysmal periods, nor the accompaniments of angina pectoris. Sir Lauder Brunton, in his well-known work on the subject, writes: "The site of the pain is usually over the sternum; sometimes at its lower, sometimes at the upper part"; . . . "sometimes it is more to the left, or may wander to the right of the sternum." At the Belfast meeting (1909) he went so far as to concede to me that perhaps the cases of sternal pain were of aortic origin, those of pain about the apex of cardiac origin. Well, my argument can afford to dispense with this equivocal fraction. Barié, and Gallavardin likewise, insist again and again on the retrosternal seat of the pain in contrast with pain "in the cardiac region." Neuburger from his very large experience emphasises the same character. Dr. Morison says the pain is sternal or retrosternal, usually about the middle of the bone. In the *Lettsomian Lectures* for 1901, Dr. Mitchell Bruce describes the "sense of oppression" as "behind the sternum." Dr. Mackenzie observes the same precision. Dr. Colbeck [1] says the pain is primarily "retro-sternal." Sir Thomas Oliver [2] well insists on the sternal site of the pain, though later he deviates into the "præcordial" phrase, without indicating any distinction between the sternal and the cardiac sites. Pawinski [3] says "the seat of anginal pain is 'der obere oder mittlere Abschnitt des Sternums,'" and he too contrasts it with the "anxietas praecordialis" of ordinary pericarditis and other heart diseases. In some cases in my notes, and in some others which I remember, the chief seat of the pain was not exactly sternal, but a little to the left of the upper part of the bone, about the second or third interspace; an obser-

[1] Colbeck, *Lancet*, March 31, 1903.
[2] Oliver, Sir T., *Lancet*, Sept. 16, 1905.
[3] Pawinski, *Deutsche Arch. f. klin. Med.* Bd. lviii.

vation which has been made by other authors; Laennec for instance speaks of the pain as sometimes seated about the left pectoralis major. The pain in the spurious form, on the other hand, is about or below the apex; a spot often marked by a persistent hyperæsthesia. In angina the hyperæsthesia, often definite enough, is present only during or just after a seizure, and is not predominant in the submammary area.

If *now we turn from the site to the degrees of the pain*, I would urge that, if in one extreme the pain is ruthless, such that the patient feels as if "the life were being squeezed out of him," if, as no other pain, it racks the very depths of anguish, and in its reiterating paroxysms of torture and dismay seems to be more than flesh and blood can bear,[1] and if in the severest cases a "status anginosus" may bait the sufferer, making him as one of the older writers says "furibundus," for hours or for days—for days in one of Heberden's cases—yet in other cases its passage may be so light and transient (a degree often called by Brera's name of "stenocardia," 1810) as only too often to escape serious attention, yet, even in these minor skirmishes, it brings with it a peculiar malice, a warning innermost touch; incomprehensible, vaguely startling. The instancy of the arrest, though but for the moment, surprises the man. A moment, and it is gone; but it comes again, and again leaves upon him this same uncanny spell; so that he goes to his physician, "just to be assured that it is nothing, nothing but a touch of flatulence, to which he is subject, as his father was before him." These slighter anginous tensions are by no means rare in cases of high arterial pressure. Did such far from uncommon forebodings begin and end just there, their identity with angina might be questioned (*vide* Diagnosis, p. 495), but we have only too much evidence of their common nature, whether as forerunners of typical attacks or as minor terms in a series of varying intensity, to doubt their sinister meaning. Allan Burns says, "Pain may not occur in either arm; it is frequently absent and not pathognomonic." As Dr. Mackenzie[2] writes: "It may be but a slight sensation of constriction across the chest . . . so slight as to pass un-

[1] *Αἰεὶ τὸ μὲν ζῇ, τὸ δὲ μεθίσταται κακόν,*
τὸ δ' ἐκπέφηνεν αὐτίκ' ἐξ ἀρχῆς νέον.

[2] Mackenzie, Jas., *Heart Diseases*, 1908, p. 44.

noticed." "If," as Sir James Goodhart[1] has said, and as Laennec said before us,[2] "these larval forms of angina, too often put off as false angina, were more seriously reckoned with, we should hear less of the rarity of the disease" (*vide* pp. 242-3). He adds these weighty words: "Angina pectoris is common among us if we are on the look out for the milder forms of it. . . . In walking up the smallest incline, the anginal patient will shy . . . stop to look into a window . . . in a few seconds the pain will have vanished . . . with nearly always an eructation of wind . . . and be regarded as a simple indigestion. . . . It finds out the city man after breakfast." Thus in his later years it "found out" an old friend of mine—a *bon vivant*, with a big heart, high pressure, and atheromatous arteries, but no renal disease; when while wielding his salmon-rod he had to lie down on the bank for a few minutes before he could go forward. Gradually the oppression became more frequent, and occasionally put on the full dress of angina; but by a careful and tranquil life he kept it at bay, and after many years of high arterial pressures died of cardiac defeat. Dr. Graham Steell[3] says: "True anginal pain need not descend into the arm, but may be confined to the chest; it may descend into the arm later." An unmarried man, æt. 34, was in the Addenbrooke's Hospital in December 1911. He had never had rheumatism in any form, nor indeed any illness till the present. Although he could give no account of syphilis, it was easy to read the story in the physical signs—aortitis, aortic valvulitis, aortic regurgitation. And he had not forgotten that, some months before, a slight but disagreeable pain—nothing comparable in degree to typical angina pectoris—had at times crossed his chest about mid-sternum, making him stop for a moment. In such instances it is that the mischievous confusion between "true angina" and "false" may betray us into grievous error, for some writers to these stealthy forms of an affliction only too real, only too ominous, give the name of "false angina." The minor attacks are quite as characteristically brought on, by effort, by cold, and the like.

[1] Goodhart, Sir Jas., *Lancet*, July 1, 1905.
[2] Laennec, *Traité d'auscultation*, 3rd ed. vol. iii. p. 351.
[3] Steell, G., *Med. Chron.*, Nov. 1913.

It is of the utmost importance then to detect, and truly to interpret, this "*petit mal*" *of angina* (*A. minor*); in themselves, indeed, these arrests are ominous, for even if, as is sometimes the case, no heavier attacks succeed them, they often indicate that high arterial pressures of long duration are straining a not immaculate aorta; and that, unless they are averted, dilatation and defeat of the heart may be inevitable, and perhaps not far off. Women often think little of some oppression of breathing on rising a hill, and, if it be unattended by any menacing sensation, they will forget to mention it. This was the case of a lady who consulted me not long ago. By no patience could I make her systolic pressure less than 180-190; and a few questions elicited a clear story of angina minor. Another lady who consulted me, in much the same condition and about the same time, did make especial mention of oppressions not more painful but in her case attended with an uncanny sensation. In many cases, perhaps in most, this first muffled aortic groan is but the prelude to the fiercer pang of stark angina. It is because its character is thus often overlooked, or its significance even denied, that one would earnestly plead for more regard to this minor form, and I would repeat that a substernal oppression of the gravest significance may yet be very slight; and that, even if not the precursor of overt angina, if not the precursor of death by cardiac inhibition in another and perhaps not severer seizure, it may nevertheless be a grave intimation or signal of Hyperpiesia. In an infectious disease it is no very rare sign of a covert aortitis.

I have been asked by more than one correspondent on this subject, if excessive tension of the ascending aorta would not suffice, without any lesion of it, to cause such a painful or "nasty" sensation. Well, one is thus tempted to think it may; but, unless the vessel were already very taut, its outer investment, which I take to be the chief source of the pain, would be less strained than the intima and musculo-elastic media, which usually are more or less in tone.[1] As with other such investments, the pleura for instance, which ordinarily on expansion give rise to no sensations, but yet when inflamed may become exquisitely painful, so in respect of the aorta I think

[1] That a slack aorta may throb painfully we are all aware.

to become painful the outer wall must be in an abnormal state. But a more effective argument is from clinical experience. If substernal tightness on exertion (angina minor) do pass off under treatment addressed to the arterial pressures, if it pass off even for three or four years, we shall yet find it prone to return; only too prone to wax into angina major, or to merge into strained and failing heart. The more one sees of these minor cases, the smaller the proportion of those which might seem to be no more than tension of a normal aorta. And, conversely, while high pressure conditions, as for instance in the Bright's diseases, are common, the proportion of these attended with stenocardia is not great. It is true that in cases of intense and universal vasomotor constriction of a passing kind, oppression in the upper thorax, transient and without ill consequences as it may be (*e.g.* p. 229), is often distressing. On the other hand, from certain casual remarks not infrequently made by elderly subjects of vascular degeneration, I suspect that fleeting anginoid sensations (without angor animi) occur more often than we realise in the atheromatous, without any graver issue so far as angina is concerned. For example:

Male, æt. 55, under my observation for nearly twenty years with aortic regurgitation of still longer standing, on a recent consultation told me that at times, especially on moving quickly, he felt "a tingling in the whole left arm," as if he had lain upon it, but more transitory.

I repeat that in my experience angina minor is not uncommon in women, in whom angina major is comparatively rare; often in them it preludes, rather than typical angina, a strain of the arterial system by high pressures, to which they as well as men are liable. I think the attacks of angina minor are brought on by exertion, however slight; not so often by ordinary degrees of emotion, nor by obscure influences moving in sleep. Sir William Osler duly notes its occurrence in both sexes, "singly or between-whiles," stopping the patient "with a nasty indescribable sensation." And Dr. Mitchell Bruce says "we must enquire for *occasional traces of anginous symptoms*; yet unless a man is stabbed and strangled almost

to death, these lighter assaults, especially in woman, *are not rarely put by under that equivocal and mischievous title* (italics mine) of pseudo-angina." Stadler also (*loc. cit.*) warns us against the possible "tragische Verlauf" of symptoms called offhand "nervous." Sir James Goodhart I have quoted to the same effect. The evil nature of this furtive pain is betrayed by the company it keeps. Physicians possessed by the "præcordial" notion are the more easily deceived, for the angina minor, except in certain epigastric cases to which I shall advert presently, is likewise almost always at mid or upper sternum, and frequently unattended by cardiac symptoms. On any such complaint then let us exact a close physical and general investigation, including estimations of blood pressure.[1] To make a diagnosis, and to communicate it in full to the patient are of course two different things; but the patient who has experienced a full attack learns only too well what gratitude may be due to the physician who had taken timely warning from the previous skirmishes.

A sure proof of the quality and malign nature of these minor attacks is to be found in their alternation with unmistakable attacks. For instance:

Male, æt. 70-80. During the first four or five years suffered from angina intense, complete, and typical; seizures at one period brought on by gentlest walking even on the level. Then recovery set in, so that he was able to discharge all the duties of a responsible position in life. To his friends he seemed to have recovered fully; but during the last three or four years on going up hills or steep stairs he has been liable again to a slight but unpleasant substernal constriction.

But we must not flatter ourselves that, in respect of life, angina minor means a petering out of the peril. A contrary example I find in the notes of a case kindly sent to me by Dr. Falkener Hill of Manchester.

Male, elderly. Arteriosclerosis noted for three or four years. In June 1908 had a typical attack of angina pectoris. Under careful

[1] Excessive arterial pressures, as the heart begins to labour, often produce a vague uneasiness or beating in the cardiac area; but in a patient of my own, a middle-aged man, long subject to an obscure disorder of the heart, falls of pressure, to which he is subject, give him "a bruised feeling about the heart."

management and tranquillity he never had another typical attack, but frequently had slight " turns "—" a substernal tightness which had been present in the typical attack but afterwards was unattended with any radiation. Yet in one of these, in the presence of friends, the heart stopped, and *death was instantaneous*."

In many cases of angina minor careful treatment and moderation of the arterial pressures, if in excess, may give the patient a respite of uncertain duration; and now and then such an one is more completely cured.

Some years ago my own attention was especially drawn to *the suprasigmoid area* by certain clinical observations which seemed to indicate that this part is *peculiarly susceptible to changes of blood pressures*; as if by its nervous endowments it might have some regulative function in this respect. I guessed that the investments of this part of the aorta were furnished with nerve end-organs, whose function it was to indicate pressure changes; so that if these bodies, and their pain columns, were submitted under disease to a morbid summation of stimuli, the corresponding spinal segment, or—in case of longer or intenser irritation—spinal segments, would be thrown into an agitation expressed, in greater and lesser degrees, as pain. Under ordinary circumstances these messages pass from point to point in silence; the reciprocating system is in continuous touch; but if in high aortic pressures the suprasigmoid parts and arch become impaired, the resentment may take the form of angina minor—a slighter lesion of the fabric of the vessel; if there be deeper lesion, then arterial fluctuations, on whatsoever planes, may throw the associated nervous centres into wider and wider circles of functional dispersion and even agonising vibration. Under these circumstances of quivering alertness however the surprise of sudden death is less frequent, for the vagus is sooner tired than the augmentors. As in light chloroform anæsthesia, it is in the startle or nip of an incipient seizure that the fully charged vagus may sever the thread of life, even before the patient is aware of his peril. Thus oftentimes it is by inference only that the name of angina may be called upon to explain the event.

Concerning *this mechanism, by which the pain is awakened*, we cannot as yet speak precisely. I had suggested that nerve end-organs, whose sensitiveness in other parts to pressure, and

especially to dragging tensions, are well known, would be discovered in the sheath of the ascending aorta. Hitherto however I had not succeeded in obtaining dissections made with this purpose; but in Dogiel's now classical papers [1] we find that these end apparatus have been found in the connective tissues of the pericardial, adventitial, and other investments about the collar of the heart and along the large vessels. The pain is of the kind which belongs to fibrous tissues. That of acute pleurisy, if less vital, less searching, yet is similar in kind. In inflamed tendinous structures, where pacinian bodies are abundant, the pain is agonising—as in acute gout. So it is again with drags on the mesentery.

Into the many records of eccentric *distributions of the pains*, which do not serve to illuminate my main purpose, I need not enter at great length. In Gibson's *Morison Lectures* [2] will be found the diagrams, to which I have referred, of these unusual distributions of pain; as, for instance, round the points of both scapulæ to a small area about the fourth thoracic spine. And these eccentricities are not merely curious; on a true perception of the meaning of an odd radiation may depend the true interpretation of a particular case. Tender cutaneous areas, such as delineated in Mackenzie's and Gibson's text-books, if perhaps capricious, and so of less practical value, have a physiological interest, to which I will return. Normally, the pain originating under the sternum radiates, as we know, in the areas which Heberden described, and which since his day have been abundantly verified. Beyond the breast, the pains are referred approximately to the large lateral branch (the intercosto-humeral) of the second thoracic, which extends outwards and backwards to the integument of the inner and posterior part of the arm (cutaneous branch of the n. brach. medialis), reaching nearly to the elbow, and towards the nerve of Wrisberg and other branches of the cervico-brachial plexus; such seems to be the content of the segmental representation. I note the intercosto-humeral reference especially, because it definitely brings the second thoracic segment into the anginous centres. The pain in the arm and hand is usually referred to the ulnar aspect (eighth cervical

[1] Dogiel, *Arch. f. micr. Anat.*, 1898, p. 66; and again 1903.
[2] Published in 1904. See also Hirschfelder, *Dis. of Heart*, 2nd ed. p. 373.

and first thoracic), but not invariably; sometimes the distribution is rather median (see my case of the whole left arm, of the left thumb and index in Bretschneider's case (p. 298), and many others). Pain referred to the outer half of the upper arm and the radial half of the forearm and part of thumb, an infrequent distribution in angina, indicates the fifth and sixth cervical segments. Pain in the trapezius and sternomastoid suggest vagus and spinal accessory centres, and—thence the second, third, and fourth cervical segments (hyperæsthesia of head and neck (Mackenzie)). In forcing attacks the pain may drive into both arms, the upper left breast, and—according to Mackenzie's shaded area—the neck, throat, larynx—especially "pomum Adami (laryngeal vagus)"—and jaw: "pallid, anxious, and *speechless*," says one observer of his patient. In one of Fothergill's cases, and many later, the pain was referred to both arms. John Hunter's attacks (auto-inoculated syphilis) began, as he carefully describes them, with a sense of tightness in the muscles of the nose, face, and left jaw; then pain passed down the arm to the thumb (not on the usual ulnar side).[1] It was retrosternal also, crushing the bone as it were to the back. He noted that the arteries of the left arm became tender, even to slight touch. He also had the throat pain; during the attack he could not swallow, and could hardly speak. He was pinched and pale in face. The aorta after death was of the (syphilitic) kind described by Hodgson (p. 168): "The ascending aorta was much thickened and dilated, and tuberculated on its inner coat." In a case by Dr. Sidney Phillips, already noted (p. 264), the pain drove itself sharply into the throat. Dr. Hugh Anderson asks if this not very uncommon direction of the pain may forebode vagus perils. Or may pain about the pomum Adami be significant only of high blood pressures? In some records we read how, in forcing attacks, and the more as the spinal segments under accumulation of stresses become unstable, pains may be distributed over fields still larger or more exorbitant, such as the hypogastrium, the tongue (Trousseau, Ebstein, and a case

[1] In respect of certain of these pains see Dr. Leonard Kidd's essay on the "Phrenic Nerve" (*loc. cit.*). The part of the phrenic in these symptoms is as yet scarcely defined.

of my own), the testicle (Laennec, and the abdominal case on p. 309); the larynx, causing aphonia;[1] areas of the scalp (Head, Mackenzie; Mackenzie says that the sterno-cleido-mastoid muscle and the trapezius, if gently pinched, may prove tender). In a case of my own (G. Y., p. 519) the patient complained of bursting pain in the lobes of the ears. These and such peculiarities may depend upon obscure furrows of structure, work, or worry in the individual; but no doubt in many cases, could we but decipher the signal, they might tell of variations in the seat of the lesion. Many or all of these peculiar symptoms, *e.g.* pain in throat, neck, lower jaw, were noted by the elder observers. Laennec's case of testicular pain was a very curious one; the radiation was wholly dextral, and sped downwards to the spermatic cord and testicle, which became tumid, and to the right leg. Furthermore, as in the case of the fourth thoracic spine just quoted, or in a supposed "gastralgia," pain at ambiguous points may peep up alone and, like the trick of the lapwing, divert the unwary from the true line of quest. When however the peculiarity is but an accentuation of some point of the ordinary area, as in the left little-finger case cited by Mackenzie, or as in a case of my own (p. 294) in which walking uphill, dancing, etc., aroused pains in the centres of the palms of both hands (7th cervical segment ?), or as in Heberden's "Unknown," in whom the pain started just above the left elbow, thence passing to the chest; or again, as in MacBride's case,[2] where in each arm the pain arose precisely at the insertion of the pronator teres on the radius—occupying a spot about the size of a crown piece, the rest of the limb being free—in these and such instances of Potain's "angine renversée" the ambiguity is not so great.

A male patient, æt. 67, sent by Dr. Dockray of Bishop's Stortford, suffering from angina pectoris, became subject, on walking up any slope, to pain about the junction of the middle and lowest third of the sternum, but also—on which he laid more stress—to a simultaneous pain around the points of both elbows. These elbow

[1] A condition which may be hard to discriminate from latent aneurysm, unless by the laryngoscope, an examination of course impossible during angina. Fothergill also noted a reduction or suspension of the voice.

[2] MacBride, *Med. Comment.* vol. v.; (and Smith) sixth volume of *Med. Observations and Enquiries.*

pains were excruciating; though often mitigated by dropping the arms, at times they would bring him in an agony almost to his knees. These parts—the sternum and elbows—were the only seats of the pain. He had no definite angor animi.

In a man æt. 58, a patient of Dr. Fordyce of Cambridge, the pain was at first subscapular, on both sides, but especially on the left; and, although before I saw him it had taken up the typical positions, substernal and left arm, yet the subscapular pains continued. The case was one of high blood pressure (pressure at our consultation at least 180 mm.). I found a systolic murmur at the base of the heart, and a thudding second sound. The pain, which coursed down the left arm to the elbow and wrist, did not reach the fingers, but they would (all) turn cold and numb. Neither then nor later did we find reason to diagnose aneurysm.

A gentleman, engaged in an anxious business, asked me to examine his heart. He was a plump and fresh-looking man about 45 years of age. He denied syphilis, and the Wa. test was not then familiarly known. His arteries appeared to be normal. I examined his heart with suspicions awakened by his own; but, finding the size of the chambers and the qualities of sounds to be normal, I assumed that he was frightened or hipped. He had no palpitation nor shortness of breath, but he asserted that there was something wrong about his heart which he seemed unable to describe. I reassured him as well as I could, and advised rest and a sea-voyage. Two or three weeks later he wrote to me (he lived far away) to say that he felt no better, that he was convinced I was wrong in my confidence; and now for the first time spoke of a pain arising in his left hand on exertion. This was alarming, and I begged him to call upon me again. On his second visit I heard from him, what before he had not thought material, that on ascents he had felt a pain in the centre of both palms, especially of the left; and latterly he had begun to associate this pain with the indescribable suspicions concerning his heart which had appeared to me to be fanciful. No more signs were to be discovered, but I sent him home with a note to a medical friend in his neighbourhood, urging immediate treatment based on the gravest view of the case. And before long the symptoms, at first so obscure, assumed the unmistakable form of classical angina; so that, instead of taking the change and rest which he had been contemplating, he was invalided at home; and in this state he remained for some time, unable to take any exertion without the imminence of an attack: for ten days indeed he was unable to leave the house. His chest was afterwards examined by an expert observer at a watering-place, who told him that, even now, no organic disease is to be discovered in heart, arteries, or elsewhere; and that, history apart, he would "seem a proper candidate

for life assurance." On his last visit he told me he had been able to take the sea-voyage with great advantage. [Some time later however angina reappeared in full dress, and he died in an attack.]

Speaking generally then, the distribution of anginal pains is, more commonly and typically, by the last one or two (7th rare) cervical and the first one or two thoracic segments, with frequent association of the third and fourth cervical, rarely of the second. The distribution to the upper cervical branches is represented in the vagus and the spinal accessory segments.[1] As the state grows worse, the pains usually occupy more of their normal province. Fierceness of assault, or storm, as in status anginosus, may signify intensity of local lesion ; but is at least as significant of harassed centres. Stimulation is cumulative ; it calls, but may be resisted, even up to 15 or 20 repetitions ; it is so stemmed that the gradient of it has to attain a certain steepness before it overflows the threshold ; then, if I may use the figure, the garrison is affrighted, defence is shaken, and agitation spreads from quarter to quarter.

Dr. Morison draws our attention to the remarkable swiftness of the vanishing of anginal pain, that is to say, in the ordinary run of cases, and in the earlier stages ; a curious feature, which may depend on tides of blood pressure. But in one definite case of angina of my own (Mrs. K., p. 242), the left arm on the contrary became the seat of pain intense and continuous, apparently not confined to particular nerve tracks, for ultimately it occupied the whole arm up to the wrist, and after fatigue or anxiety would become for many hours, or a day or two, so agonising and persistent as to drive away sleep. The patient was indeed far more distressed by the arm pain than by the "stenocardia," which in this case never attained any great severity, and was evoked only by walking upwards. While the patient at rest might be suffering much from the arm, the stenocardia was often absent ; though the connection of the pains was only too certain. Suspicions, or hopes, of neuritis led again and again to re-examinations of the arm, which however, except in superficial and transient degrees after an attack of pain, was at no points tender to pressure ; and the aspect and nutrition of the arm and its

[1] M'Kendrick, J. S., *Glasgow Med. Journ.*, Dec. 1909.

muscles were in no wise altered. In this case angina pectoris gradually became more and more severe and typical, till death in a seizure. In a syphilitic case in a comparatively young man (age about 30 to 35), the angina began not as pain but as discomfort or sense of weakness in the arms; in later attacks however pain (in both arms) was brought on at once by ascents; but soon it occurred during rest also, and became terrible. One or two of his worst seizures awoke him after midnight. Either he had little chest pain, or in the agony of the arms he forgot it (see case, p. 293). But at times the pain took the ordinary form, including retrosternal distress, and it was in one of these more typical attacks that he died. I fancied that in the exclusively arm variety the vagus might not be affected, but I have an instance of no such immunity. We cannot then be too suspicious of pains uniformly recurring on bodily exertion, however eccentric their seat; especially if in elderly or possibly syphilitic persons.

Two more cases of eccentricity I have deferred because they lead to another side of my argument; cases in which the pain was most intense in the belt of the eighth and ninth ribs. In both the pain had been described, according to custom, as "in the præcordial region"; but, being sceptical about this phrase and desiring data more precise, I perceived in each case that the patient put the points of two fingers far below the heart, namely, on the eighth and ninth ribs in the anterior axillary line, and towards the costal arch. Dr. Head[1] says the pain may be referred even to the eighth and ninth dorsal nerves. One of these cases was in an elderly man with aortic atheroma, but with no evidence of mitral disease; the other in a man of 45, also with aortic disease, but consequent upon rheumatic fever. In both the pain was very intense, and more distressing from its association with cardio-arterial disease. In one it was attended with the dread peculiar to the typical malady; in the other perhaps not, but it is not easy from the hospital patient to obtain discriminating answers. In neither was the pain substernal, but in both it radiated up the anterior axillary line to the shoulder and down the left arm, so that no more could be said of the mere pain than may be said of

[1] Head, H., *Brain*, vol. xvi.

any pains sweeping the chords of the brachial plexus, whatever their origin. My point is that even in these patients the pain, although undoubtedly anginous, was not " at the heart " nor " in the cardiac area " ; it was far below it. The dart of the pain this way or that may be determined by some local condition, as in a case of Dr. Mackenzie's, in which anginal pains darted into the lower jaw, where were two decayed and tender teeth ;[1] or the sensory centres fretted by more than one eccentric provocation may be doubly irritable. Dr. Head sums up the anatomical lines, tentatively, thus :—Transverse arch, inferior laryngeal segment ; ascending arch, third and fourth cervical, and first, second, and third thoracic ; ventricles, second, third, fourth, and fifth thoracic—corresponding to the 2nd-7th rib, inner surface of upper arm, and ulnar surface of forearm[2] (I think there may be some merging here of the aortic and left ventricular areas) ; auricles, fifth, sixth, seventh, eighth thoracic, to which must be added the vago-spinal strands (" bulbar autonomic ") which carry afferent fibres. But there is much baffling overlap of areas. The auricles in their development are posterior to the ventriculo-aortic parts (more caudal). The spinal and cerebral paths are unknown. Pain may be referred from the descending aorta to surfaces below the nipple and to the upper abdomen. On clinical experience I cannot take the innervation of the heart and of the aorta to be quite identical. And there is much in these diagrams as yet hypothetical ; Dr. Otto May,[3] for instance, has published three mitral cases with " aortic " areas of pain ; and, as Dr. Beddard has remarked, physiologists in discussing these problems are tempted to argue in a circle.

Finally, the pain of angina pectoris is generally paroxysmal, rarely a " steady grind." The reason of this is not obvious. Even in the " status anginosus " flashes of pain are continually smiting across the storm. A continuous thoracic pain, however urgent, if of vascular origin at all, signifies rather some accident to the heart itself ; such as a parietal rupture, or an

[1] Mackenzie, Jas., *Heart*, vol. ii., May 1911.

[2] See also Mackenzie, Jas., *Diseases of Heart*, 1908, where he explains how as the upper limbs bud out from the trunk portions of the cervical and upper dorsal nerves are distributed to these areas of the arms.

[3] May, O., *Brit. Med. Journ.*, Jan. 1, 1910.

acute coronary thrombosis. But if, and how, severe pain may originate in the heart itself is a question to be considered presently (p. 440).

On the *paræsthetic and even paretic affections of the arm* in radiating cases, or possibly in cases without pain, I have nothing, except a few curious records, to add to what is well known to students of angina pectoris. In an arteriosclerotic case of Bretschneider's,[1] angina, with spasmodic pains radiating from the angles of both lower jaws to the points of the shoulders, and thence down the left upper- and fore-arm to the thumb and index finger (p. 292), led to an almost complete loss of power in the whole musculature of this arm; due probably to exhaustion of the segmental cells. The reporter correctly explained these symptoms as arising *from the arch of the aorta* ("von sklerotisch erkrankten Aortenbogen"), by way of reflex from the third and fourth cervical and the first dorsal segment. A patient of mine, æt. 51, who, though a teetotaller, had had many attacks of gout, and whose father was gouty and "died of atheroma," sought advice for well-marked angina minor. His systolic blood pressures were over 200. The physical signs were those of dilated aorta. Although there were no pains in them, both arms (during the seizures) "became useless." Such was the symptom of "sinistri brachii stupor" in a case of typical angina in a woman described by Morgagni (p. 283). Heberden too said that in some cases the arm is benumbed. As with the pain, the distribution of the paræsthesia is mainly ulnar, the median area being generally free, or but slightly and partially included; though, as in the patient mentioned on p. 338, all the fingers turned numb and cold. A white arm, "dead fingers," gooseskin, and the like are not very uncommon. I have mentioned (p. 288) my patient with aortic insufficiency without angina who, in the later time of his life, told me that in certain positions of the body a sense of numbness would pass over his left arm—the whole arm and hand; but, being so transient, it could not be tested, nor even exactly described. In a case recorded by Dr. Warton of Redditch,[2] after the attack, even so long as twenty minutes after (compare my

[1] Bretschneider, *Berl. klin. Wochenschr.*, 1911, No. 19.
[2] Warton, *Lancet*, April 16, 1898.

case of not simultaneous sweating, p. 339), the extreme tip of the middle finger would turn numb and pale, whence blanching and complete loss of sensation would spread gradually up the finger, which, unless restored by warmth and friction, would remain for half an hour dead and cold. The ring-finger also was affected occasionally. In Charles Sumner's case the attacks would leave the left arm numb and almost palsied for hours afterwards.[1] It has been alleged that in angina the left radial may shrink, while the right remains unaffected, but such records are not beyond question. That the pupils may alter in diameter, with perhaps some protrusion of eye-ball and elevation of upper lid, are points of some interest; in severe seizures the pupils are usually dilated, especially, it is said, on the painful side (Gibson); but in fear or pain of any kind a symmetrical dilatation of the pupils is too frequent to permit us to attribute to it a particular significance. Dr. Mackenzie says that by chafing under the nipple variations of diameter of the pupils, and gooseskin, can be elicited. To some of these signs I may return when discussing the nature of anginal pain. In the one case we are observing a direct paræsthesia, in the other a vaso-motor constriction. The vasodilators of the arm pass through the posterior roots of the sixth and seventh cervical and first and second thoracic segments.

Not a few cases are on record, in genuine angina pectoris, of outbreaks of *herpes* on the painful arm. In the case of a lady who arrived at my house after an attack, the left arm had to be gently borne on a pillow by her maid as she passed from the carriage to the house. Burns says, "In Mr. Hunter the left arm could not bear to be touched." Two physicians[2] have described a case of angina pectoris in which an eruption of *lichen planus* broke out on the inner aspect of the left arm, along the distribution of the intercosto-humeral nerve; in one of them was also a small patch on its intercostal anastomoses, above and outside the left nipple. The whole of the same

[1] Johnson, J. T., of Washington, *Boston Med. and Surg. Journ.*, Oct. 15, 1874.

[2] Gasne and Chiray at the meeting of the Société Médicale des Hôpitaux de Paris, March 31, 1905.

area was tender to touch and heat. But a doubt arises whether some of these cases of trophic or paretic affection of the arm were cases not of mere angina, but of mixed origin ; as of aneurysm or intrathoracic growth ? We know that herpes of the arm may arise from aneurysm or new growth, apparently by irritation in the course of the nerves. In one of my own cases, and in one among those provided for us in an R.A.M.C. examination, herpes had thus broken out in the line of radiation ; and Sir W. Osler had published a case of the kind.[1] In a certain intensely severe case, of my own, of anginal pain in the left arm the limb more than once became largely œdematous ; and on one occasion I witnessed this effect. Gibson, Mackenzie, Head, and others have given a good account of such phenomena ; and of graver trophic effects, such as atrophy of the arm (Gibson, Eichhorst), in Eichhorst's case with the reaction of degeneration ; to these I can add little or nothing. In one of Dr. Windle's cases [2] the left fingers were flexed and rigid. The hand may be white or cyanotic.

Characteristic "*tender points*," in my experience, as in that of Fränkel and others, are often wanting, and generally inconstant. For a short time after the attack the muscles of the area affected may be tender, all over or at particular spots ; but, as these tender spots or areas are thus inconstant, and the sensation fleeting, they have not been fully verified ; unless perhaps for the left pectoral and brachial areas. Dr. Head thinks that a hyperæsthesia of the upper part of the chest, front and back, towards the left side and left arm (second-fourth thoracic segments, and often third-fourth cervical) is often found in aortic insufficiency, and disappears on mitral regurgitation (see p. 389). The frequent absences of this reaction Mackenzie explains by the reasonable assumption that a certain height of excitement of the corresponding spinal centres is required, when a very slight additional factor may determine it. These hyperæsthetic areas on the chest, etc., had been picked out by many of the earlier writers on angina : *e.g.* Desportes, Jurine, Laennec.

To "*dextral*" *cases* I must make some allusion, if to little purpose. Wall described dextral radiation. The pre-

[1] *Lancet*, April 9, 1910.
[2] Windle, D., *Lancet*, May 13, 1911.

sumption of Gibson, Morison, Schmoll of San Francisco,[1] and others, that these are cases of right-sided heart-disease, is not yet fortified by conclusive evidence; the distribution of the cardiac nerves, vagus, spinal accessories, and right and left sympathetic, between the parts of the heart, *e.g.* rightwards or leftwards respectively, is little known. The septum is of course a late development. In many cases at any rate dextral radiation seems to represent only a more violent storm, a more forcible extension of the primary radiations. For, as I have read often and seen more than once, in the same case the pain in one attack may be confined to the left side; but in the next, if more severe, or quickly following, it may cross over. On glancing through my own cases I am surprised to find in how many dextral radiations occurred, more or less. I find only one in which they were dextral only, and in this case no necropsy was obtained. Still, there are not a few records of cases consistently dextral, and dextral only. One by Dr. Morison [2] was as follows:

Male, æt. 29. Malignant endocarditis. Severe attacks of pain "over the heart," passing up to the right shoulder and down the right arm, being particularly felt at the tip of the right little finger. Also frequently acute and constricting at the lower end of the sternum. There was a double thrill on palpation over the second and third left rib cartilages, and a harsh double murmur there; also a systolic murmur at the apex—not traceable to the left. P.M. Gross lesions of the pulmonary valve, *the base of the pulmonary artery* (italics mine), and the septal wall of the right auricle. Aortic side fairly free from disease.

Even if a right-sided angina were attributable to some lesion of the pulmonary artery near its exit, yet it is not easy to see how the seat of origin of the irritation, if virtually within the heart itself, could have much influence in determining the range of the radiations to right or left; if it be outside the heart and in the pulmonary artery, then sinistral angina is of aortic origin. Dr. Morison includes his case of dextral angina among the effects of dilatation of the right ventricle; a common event, of which such pain is no ordinary sign. Dr. Sidney Phillips also, starting

[1] Schmoll, *Münch. med. Wochenschr.*, 1907, No. 41, see p. 353 note.
[2] Morison, A., *Trans. Path. Soc.*, 1872, and *Lancet*, Jan. 8, 1910.

according to custom from the assumption that strain of the left ventricle is the cause of sinistral angina, is of opinion that dextral angina pectoris may result from affection of the right ventricle. When, as occasionally happens, the right-sided radiation is primary, and even exclusive, there must be some determining factor of this distribution.[1] Dr. Byrom Bramwell suggests that pain in the right arm may point to the aorta, in the left to the heart.

But evidence of another kind, published by Dr. Drummond of Newcastle, may have more bearing on this problem. In a very interesting study of aortic aneurysm,[2] illustrated by diagrams, Dr. Drummond found that when this affection is associated with angina pectoris the radiation is usually to the left side, but sometimes to the right. He then believed himself able to formulate certain relations between the distribution of referred pain and the site of the sac; the rule seemed to be that a sac springing from the anterior aspect of the arch was associated with pain referred to the left arm rather than the right, whereas the nearer the sac approached the upper and posterior aspect of the inside of the transverse portion of the arch the greater the tendency of the pain to pass to the right shoulder and arm. Sacs originating beyond the subclavian artery do not produce pain in the arms. "One of the most interesting and constant observations," he adds, "was the connection between the root of the innominate and pain at the back of neck and occiput." Dr. Drummond tells me however that two ambiguities in the records made his results less trustworthy; first, that of defective clinical records, and, secondly, in the case of the larger sacs, that of determining from post-mortem appearances the point of the arch from which the sac really sprang. An interesting case, in some corroboration of Drummond's opinion, was quoted by Dr. H. B. McCaskie in his M.D. thesis.[3] In a patient suffering from angina, the pain radiated down the right arm. Now on autopsy an aneurysm was found on the ascending arch; but there was also a second one, and this arose from

[1] Vide *Brit. Med. Journ.*, June 13, 1908.

[2] Drummond, D., "Thoracic Aneurysm," *Brit. Med. Journ.*, June 13, 1908.

[3] McCaskie, thesis for M.D., Cambridge, March 6, 1909.

the summit of the arch. Dr. Bradbury and Dr. Wright read notes and exhibited the parts to the Cambridge Medical Society, on November 3, 1911, of a case of rupture of the aorta in a man æt. 53. The rupture took place on the descending arch; the severe paroxysm of pain, "like angina pectoris," which attended it was referred to midsternum. The whole arch however was very atheromatous. Radiography may produce evidence for us on this part of the problem. I see that Neuburger had an autopsy in a dextral case, but he offers no explanation of the aberrancy. With the customary prepossession he seems to have confined his attention to the heart and the coronary arteries. (See also p. 421.)

That left and right angina depend on disease of the left and right coronary arteries respectively, and their relations to the cardiac plexuses, is a fanciful notion which has been disproved by many observations.[1] For my part I repeat that severity of the pain has much to do with this cross over to the right. A patient of mine, a lady who suffered by ill-hap from syphilitic angina, told me that when the pain was at its worst it would pass over to the right arm also.

Some interesting *observations upon the courses of aortic pain* were published, also in an M.D. thesis at Cambridge, by Dr. Willoughby,[2] who investigated embryological evidence of referred pain. Beyond the innominate, the origin of the arch rises entirely from left-sided vascular arches, which may imply referred pain on the left side. The first portion of the arch originates in a fusion of two symmetrical right and left ventral vessels, so that it would be impossible to say how far pain from thence should be distributed right and left respectively; especially if the seat of the lesion on the circumference were not precisely ascertained. Dr. Willoughby accepted my attribution [3] of angina pectoris to lesion of the arch of the aorta, and considered that this view "broadened the horizon of the etiology of the disease." The distribution of atheroma did not seem to him to betray any genetic lines of incidence. Embryologically

[1] *E.g.* von Neusser, *Clinical Treatises on Disorders of Respiration and Circulation*, Part III., Eng. trans., New York, 1909, p. 13.

[2] Willoughby, W. M., reported in the *Lancet*, April 16, 1904.

[3] In my *Lane and Cavendish Lectures*, and earlier and later papers.

speaking, he gathered that pain taking its rise from the supra-sigmoid part of the aorta might be bilateral; that it would be referred to the region about the junction of the manubrium with the body of the sternum, and radiate probably to left, or to both left and right. The left ventricle, being wholly formed from left-sided embryological parts, should refer its pain entirely to the left. On the whole, he thought the predominant bent to left-sided radiation of anginal pain to be in accordance with the preponderating left embryonic origin of the seat of the causative lesion. As regards left-sided radiations, some anatomists describe the anastomoses of the cardiac nerves, and the corresponding spinal segments, as closer on the left than on the right side; if so, it would be easy to understand how slight individual variations in this construction might vary the quarters of radiation.

In the earlier attacks of a series, the pain usually ends with surprising promptitude; the patient passes from hell into heaven; but as controls weaken, and centres by summation of stimuli are fretted and exhausted, the relief comes more slowly, and is less complete; the patient is left tremulous and enfeebled.

It is of more practical importance to turn to a grave and deceitful class of rarer cases; namely, the *epigastric or abdominal angina*—"præcordial" in a more proper sense of this ill-managed word. It seems that these cases, long recognised by students of atherosclerosis and angina, are not yet familiarly known in general practice.[1] Yet Heberden did not fail to describe this distribution of the pain: "alii alterio ex abdomine orientem, tumque demum pectus corripientem."[2] Von Dusch recognised this epigastric variety of angina, and stated that the pain, or peculiar uneasiness, referred to this region, may radiate backwards to the spine; as it did in Brunton and Williams' case (p. 309). Butter,[3] while describing the usual seat of the pain as mid-sternal, says it may arise occasionally at the pit of the stomach. But Butter is not so accurate a writer as Heberden or Parry, and seems to mix up with angina

[1] See Discussion in the *Lancet*, May 18 and June 1912, where more than one eminent speaker referred to abdominal angina as a new clinical discovery. It is of more frequent occurrence than those speakers then supposed; and has long been familiar at any rate to French and German physicians.

[2] Heberden, *Edin. Med. Comment.* vol. ix. p. 307.

[3] Butter, *On the Disease commonly called Angina Pectoris*, London, 1791.

different kinds of distress, such as "gout of the stomach" and other cardiogastric perturbations. In a letter to the *Lancet*, on one of the dates mentioned, Sir W. Osler attributed to Leared the honour of the first description of "Angina abdominalis" as a group of cases. To Potain these cases were well known in the vessel as "angina subdiaphragmatica"; to Huchard and Neusser as "angina pseudogastralgica," and later, under names even more complicated, to Ortner, Hasenfeld, Pal, and others. K. Müller of Budapest described it as angina abdominalis, and dwelt on the difficulty of diagnosis. There is no doubt nevertheless that many kinds of disorder have been indiscriminately gathered under this last equivocal title.

More than twenty years ago, with Professor Bradbury, I visited Professor X., a man of sixty years or more, with degenerate arteries, who, after hastening upstairs with a small portmanteau in his hand in a foreign hotel, had felt rather distressed, and began to complain of recurring attacks of "indigestion." We saw him soon after his return home. He assured us that if he could but get rid of a "nasty feeling of imprisoned flatulence at the pit of the stomach, he would soon be all right." But as the uneasiness—for, importunate as it was, it scarcely could be called severe pain—was recalled by walking, and carried with it a subjective sense of indefinite apprehension, Professor Bradbury's fears were aroused, and a consultation was held. In view of all the phenomena, we agreed without hesitation that the case was one of epigastric angina pectoris, and, for the moment, we hoped in a manageable degree; but the patient grew worse, and within a few days died in an attack. Romberg reports a case of this distribution in which the pain in the same area was more severe, so severe that the first notable seizure was momentarily mistaken for perforation of a gastric ulcer. Another case, in which the epigastric uneasiness was little more than a vague subjective sense of oppression and distension, was in a Mr. A., an elderly man with degenerate arteries and enfeebled heart whom in my Leeds days I often saw with Dr. Clarke of Doncaster. This discomfort, and indeed the sudden death which took place later, were not inconsistent of course with mere cardiac decay; but our suspicions of epigastric angina were aroused in part by the general features of the case, but especially by two

facts observed by Dr. Clarke, that the attacks were brought on by exertion, rising to the night-stool, or contact with cold bed-clothes; and that they were readily subdued by nitrites. And in the subsequent course of the case his diagnosis seemed to be verified. I would remind the reader that the connective tissue about the pancreas and solar plexus and the adventitia of the larger blood vessels of the part are rich in Pacinian bodies.[1] Enterospasm or any drag upon the mesentery thus endowed may give rise to agonising pain. We shall agree that disease of the aorta, and possibly of other large abdominal vessels, may make itself felt after the fashion of angina; but paroxysmal pain alone is no criterion.

Clinical observation suggests that epigastric angina may come of a thoracic lesion, for some obscure reason referred now to the breast, now, even in the same patient, to the abdomen. In Dr. Glasier's case, mentioned on p. 149 of this book, the patient's first attack, or attacks, were epigastric; afterwards they settled wholly in the breast, and remained there. Dr. Johnson Smith [2] also has related a case in which the attacks alternated between the chest and the abdomen. In one of the cases published in Sir W. Osler's *Lumleian Lectures* a man, æt. 59, had both ordinary (typical) attacks as well as attacks of the epigastric form. He died in one of the ordinary attacks. Osler agrees with other authors that the pain in the upper abdomen may "rise up"; so that, as one patient put it, "when it gets beneath the breast-bone, it cuts me short as if the machinery of life had stopped." Pal's case (recorded in his *Gefässkrisen*) moved in like manner from abdomen to chest. But long before these Hamilton [3] had published a case of this metastasis of the pain from abdomen to chest. In some cases of coronary thrombosis (cf. Morgagni's "Medicus, æt. 62") the pain has thus started from below and ascended from the abdomen to the thorax (p. 311). Of such a case I owe a report to the kindness of Dr. Campbell Smith of Tunbridge Wells.

An elderly man had been subject occasionally for eight or ten years to symptoms of doubtful interpretation, whether of dyspeptic

[1] *Vide* p. 421, and also Ceelen, *Virchow's Arch.*, June 11, 1912.
[2] Smith, Johnson, *Lancet*, May 18, 1912.
[3] Hamilton, *Med. Comment.*, Edin., vol. ix. p. 307.

or of cardiac origin. But he had had long intervals (even two years) of freedom. No gout, no lead, no tobacco, no alcohol; but a sedentary life. In 1910 began definite attacks of angina in the epigastrium—not radiating towards the heart or elsewhere. He would sit up, and take nitroglycerine tablets in large numbers—on some days as many as eighteen or twenty, usually six or seven, and one or two inhalations of amyl nitrite. As a rule, no alteration in any quality of the pulse could be felt by the finger, but sometimes the pressure seemed to rise. His systolic pressure at rest was usually 150. The urine was quite normal, both as to constituents and the proportions of its solid contents. In the later stage of his illness, however, the pulse became "weak and irregular"; and on one of these occasions it was that he died suddenly.

Vaquez and Bordet,[1] again, describe a case of unquestionable angina, starting from the epigastrium, and afterwards seating itself behind the sternum, thus:

Mons. X., æt. 55, had suffered for six months from typical angina pectoris. The crises started at the epigastrium, but then mounted up "behind the sternum," taking the usual course. Percussion and auscultation were negative; but not so the orthodiagram: this revealed "une altération très prononcée du vaisseau" (*i.e.* of the thoracic portion of the aorta).

So that epigastric angina does not necessarily signify a subdiaphragmatic lesion of the aorta.

The absence of dulness over the aortic region they explained by the dilatation—to almost double its normal diameter—not at the "cross," where it nears the surface, but at the ascending portion of the vessel. The authors are of opinion that in high-pressure cases the dilatation is chiefly to be found at the "cross." This is not my experience; I have often seen it there in many cases, especially in Hodgson's aortitis, with pressures no more than ordinary; as also in cases of aortitis in which the tone of the vessel was much slackened, as it is liable then to become. In such cases the vessel may rise and fall, as in Minet's case, p. 309. Edgren has given a clear description of these epigastric and abdominal varieties of angina; William Russell likewise, Stengel, and other authors; Neuburger again has met with a few definite and unmistakably genuine instances; so that the variety

[1] Vaquez et Bordet, "La Radiologie dans les aortites," *Paris méd.*, juillet 1911.

is really fairly well established and defined. When this pain, as in many of these cases, is seated at the ensiform cartilage it falls geographically within the sternal district. Of Breuer's two cases of "abdominal angina"[1] the pain in the first was at the cartilage:

Male, æt. 40. Fearful pain at ensiform cartilage, brought on or aggravated by meals or exertion, especially on ascents, compelling him to stop "as if an iron fist were crushing his stomach" (Magen). "At the time is pallid, speechless, and anxious. One of the attacks began during the night in sleep. He was relieved by half-drachm doses of diuretin."

Relief by nitrites is often mentioned as a criterion of diagnosis, but nitrites may relieve a gastro-intestinal colic. The abdominal crises of tabes however are not relieved by nitrites, so far as I know. Dr. Somers of Selby, of a published case of his own, said that in the abdominal attacks the pulse was never appreciably affected. Dr. Walter Verdon[2] explains these cases as a cardiospasm of the stomach, a suggestion to which I shall return. Other physicians have explained them, not as angina, but as an, ill called,[3] "dyspragia intermittens," with morbid irritability of a stomach atrophied by arteriosclerosis, an opinion more opaque than Dr. Verdon's. F. R. Miller[4] of Toronto points out the different effects on different animals of irritations of stomach or œsophagus. Dogs, cats, and rabbits differ thus. In rabbits, which cannot vomit, the blood pressure rises; the contrary is seen in cats.

I believe then that epigastric angina is but a somewhat aberrant mode of ordinary angina, and due, broadly speaking, to disease of some part of the thoracic aorta; but it is not easy to detect the conditions which determine the pain to the abdomen. Can it be diaphragmatic in origin? The diaphragm is innervated by pairs of spinal nerves, besides the phrenic; and the muscle was originally a circulatory mechanism, its respiratory function developing later. Vaquez and Bordet give good grounds for hope that radioscopy will serve to indicate

[1] Breuer, "Zur Ther. u. Pathogn. der Stenokardie etc.," *Münch. med. Wochenschr.*, 1902 (No. 39 *et seq.*).
[2] Verdon, Walter, *Lancet*, June 8, 1912. [3] Dyspragia means ill luck.
[4] Miller, F. R., *Pflüger's Arch.* vol. cxliii., 1912.

the seats of many of these latent aortic lesions, acute and chronic. (For Diagnosis see p. 500.)

Angina more definitely abdominal may be an event of disease in the lower portion of the aorta or in other large vessels there. In a case recorded by Max Buch,[1] the pain was in the upper abdomen; it was much intensified by exertion, and accompanied with great tenderness of the abdominal aorta. After death a cirsoid aneurysm and highly atheromatous abdominal aorta were revealed.

The following very interesting case was recorded by Minet:[2]

Male, æt. 27. On the seventh day after defervescence from scarlet fever the temperature rose to 100·4, with abdominal pains. He was sleepless and had cold sweats. Pains grew worse, and pulsation in abdomen was seen and felt in time with the pulse, and became "enormous." The pains also became agonising and struck into the testicles. The abdominal aorta was tender to the touch, enlarged, and curved, with concavity to right. No albumen, nor other suggestion of renal disease. The symptoms slowly subsided, and the case ended in complete recovery.

A clearer proof of (syphilitic) disease of the abdominal aorta was published recently by Nixon and Walker Hall[3]:

Girl, æt. 20. Congenital syphilis. In the presence of the physicians an abdominal aneurysm burst suddenly, causing a horrible agony of pain, due no doubt to the sudden stretching of the investing tissues there so rich in sensory end-organs. The whole length of the aorta was diseased; the adventitia was abnormally thick and dense, full of cell-nests, and the vasa vasorum much diseased. Much small-cell infiltration in the media.

In Breuer's second case (*loc. cit.*), of syphilitic origin, the pain was definitely below the xiphoid and epigastrium. In a man æt. 68, Brunton and Williams[4] reported that the pain struck from the navel to the back, and thence to the chest. The heart seemed normal, and the blood pressure was normal.

[1] Quoted Stengel, *Brit. Med. Journ.*, Oct. 20, 1906. In Stengel's reference list, Max Buch, *Progressive Medicine*, Dec. 1905.

[2] At a meeting of the Soc. Méd. des Hôp. de Paris, on July 12, 1912. Quoted in the *Lancet*, August 10, 1912.

[3] Nixon and Walker Hall, *St. Barth. Hosp. Reports*, vol. xlvii., 1911.

[4] Brunton and Williams, *Lancet*, April 1912, p. 921.

The attacks were started by effort and relieved by trinitrine. A necropsy was not obtained.

Max Buch surmised that atheroma of *the mesenteric vessels* might give rise to soreness, paroxysmal colic, windy distension, and oppression; and of late under this name of Abdominal Angina many such cases have been described and discussed (*vide* essay on Atherosclerosis, p. 445), first perhaps by Schnitzler; but the name is often misleading, often the symptoms have not the definite form of angina, nor yield, or not distinctively, to nitrites. Perutz [1] however, in discussing these abdominal symptoms in a case in which atheroma of the thoracic aorta and of the coronary arteries were also present, attributed them to sclerosis of the abdominal aorta. When, as in a certain syphilitic case, there was dyspnea, we may presume that the mischief had gone beyond the conditions of angina. The often repeated experiment, originally Bernstein's, of irritation of the sympathetic in the abdomen, which, unless both vagi are cut, stops the heart in diastole, may throw some light on the mechanism of death, but does not locate the pain. Moreover the phrenic nerve is linked to the cervical nerves, and these again to the cardiac. But I find that Haddon's well-known case of angina,[2] in which the phrenic nerve was involved in an enlarged gland, was complicated with an "atheromatous" and aneurysmal aorta (syphilis ?). Many authors attribute the pain in these cases to the sympathetic branches and ganglia,[3] but it is fairly certain that these ganglia or fibres are not conductors of pain. I suggest that the pain arises by irritation of sensory nerve-endings in the investments of this large vessel. However, the obscurity of the origin of such pains, and the ambiguity of using for them the name angina, makes us uncertain whether the paroxysms are aroused in the vessels themselves (adventitia) or in the highly sensitive mesentery pulled upon by windy or other commotions. I have argued already (p. 445) that cases of many origins are confused under the accommodating name of abdominal angina. However, as Breitmann [4] says, in patho-

[1] Perutz, *Münch. med. Wochenschr.*, May 22 and June 5, 1907.
[2] Haddon, *Edin. Med. Journ.*, July 1870.
[3] See Pal, *Wien. med. Presse*, quoted Lubarsch's *Ergebnisse*, 1910.
[4] Breitmann, M. J., in a Russian journal, abstracted by the author himself at sufficient length in *Zentralbl. f. Herz- u. Gef.-krankheiten*, Dec. 15, 1913.

genesis some cases of abdominal angina very closely resemble the thoracic. But, if so, may I ask, Where is the inevitable heart factor in this variety ?

Breitmann agrees that abdominal angina is rare in women, and that many cases are syphilitic. This form prevails between the ages of 40 and 50, but a few of his syphilitic occurred æt. 20-25. He puts all down to the arterial disease, and does not see that stretching the investments may cause the pain in the aortic disease, in the thrombotic, and in other accidents in the same region which simulate it. Breitmann's list is a heterogeneous collection, and seems to contain thrombotic cases (he refers the melæna to vasodilatation !), gall-stone, and other colics ; tabes is deliberately included as "anginoid" ! His diagnoses of leading cases are not by any means always confirmed by necropsies. Clinical and pathological precision has yet to contribute to a better analysis of these phenomena.

The pressure pains of aneurysm are not very likely to be confused with these anginous symptoms (see Diagnosis, p. 498). In Sir William Osler's *Lecture on Aneurysm of the Descending Aorta*,[1] neither in the case histories nor in the summary are anginous pains mentioned. I have heard it said that aneurysm of the abdominal aorta may be attended with symptoms of anginiform character ; still it is noteworthy that in abdominal aneurysm definite reports of anginous symptoms, or sudden death, are hard to find. The pain of embolism of the mesenteric arteries has never, I think, been taken for angina ? For these reasons I am disposed to think that "epigastric angina" at any rate springs from lesion of the thoracic aorta, but, after a manner not unfamiliar in other cases of the kind, it is by some unknown interference shunted on another line.

Furthermore, in Osler's article on Angina and Aneurysm,[2] a patient suffered for six months from attacks of abdominal pain, attributed to colic or flatulency, but ultimately traced to an aneurysm of the *thoracic* aorta. In some other cases the seat of angina, at first abdominal, has afterwards settled in the usual sternal position. On the other hand, it is true that in some of these cases of paroxysmal pain, and death by inhibition, in which the pain was seated below the epigastrium, atheromatous disease

[1] No date or place on my reprint of it.
[2] Osler, *Medical Chronicle*, May 1906.

has been discovered in the abdominal aorta or cœliac axis ; but atheroma of the abdominal aorta is common enough, and careful comparisons with possible disease elsewhere are not recorded. However Barié and Colombe have published two very significant cases,[1] for in both extensive disease of the abdominal aorta was disclosed at the necropsy ; in one of them the cœliac axis was almost occluded. Teissier's test for angina of abdominal origin of excessive pressure in the dorsalis pedis is in more than one respect fallacious. In the discussion[2] on Brunton and Williams' case subsequent speakers seem to have mixed up many kinds of irregular spasmodic abdominal pains with those of Sir Lauder's text ; still some were mentioned in which atheroma of the abdominal aorta and cœliac axis were discovered after death. But again no careful comparison of states of other portions of these vessels was recorded. Albu[3] says that in these abdominal cases, if the patient be thin, the thickened arteries may be palpable ; he remarks on the combination with angina of the breast, but thinks it is rare. As differential features from colic, he gives short duration, complete independence of meals, and arousing of the pain by bodily effort.

Dyspnea.—It is agreed among careful observers that in mere angina there is no dyspnea. Records to the contrary are, generally speaking, to be counted as errors or shortcomings in diagnosis. A desire for air there may be, but the respiratory movements, if quickened at all, are shallow, weak, and not urgent. Seneca (Ep. 54), who described his angina as "brevis autem valde et procellae similis impetus," did indeed use the word *suspirium*, but probably meant no more than a sigh. The voice, if speech be possible, is hollow and distant. In his article on Angina sine dolore, Dr. W. W. Kerr[4] has not avoided this ambiguity. Some of his cases are no more than the ordinary recurrent attacks, often nocturnal, of cardiac dyspnea. Morgagni[5] acutely commented on this inexplicable absence both of cardiac disturbance and of dyspnea, as on the sufficiency of

[1] B. and C., B. and M., Soc. Méd. des Hôp. de Paris, Feb. 1913.
[2] *Proc. Roy. Soc. Med.*, April 1912, p. 53.
[3] Albu, "Diag. d. abdom. Krämpfzustande," *Deutsche med. Wochenschr.*, 1912, No. 22.
[4] Kerr, W. W., *Journ. Amer. Med. Assoc.*, April and May 1910.
[5] *De Sedibus*, Ep. xxiii. (8), "De palpitatione et dolore cordis."

the ventricular function; and to this effect quoted and compared cases from Cowper, Haller, Vieussens, and other authors. Desportes says emphatically that neither in pulse nor respiration are generic characters of angina to be found. If, he says, dyspnea be present, the case is mixed with cardiac, pulmonary, or renal disease. Usually it signifies that the heart is succumbing to high pressures, or with intrinsic disease. Allan Burns says clearly (p. 140, ed. 1809): "If there be actual dyspnœa present you may be certain that the disease is either not Syncope anginosa or that it is a complicated case—such as effusion into the chest, ossification of the valves, or asthma." He cites John Hunter's personal testimony to this effect. For good records of such "mixed cases," I may refer to the interesting paper by Dr. Davenport Windle[1] on angina coincident with cardio-arterial disease, pulsus alternans, and dyspnea chiefly of the paroxysmal high-pressure kind; but, as Heberden says of one of his patients, "adeo ut cordis et pulmonum negotium plane cessare sibi videretur," and adds, as a general maxim, "nulla tenentur spirandi difficultate." This to the elder writers, who did not always discriminate between angina and asthma, was one of its strange ambiguities; "quod de nullo alio asthmate, ut videtur, valet." "Sine quavis tamen dyspnœa," declares Willis. "If he breathes," said John Hunter, "it is by an effort of the will." Parry and Wichmann also speak no less definitely of the exclusion of dyspnea from the characters of angina; and Jurine says, "l'acte respiratoire continue à s'exercer . . . n'est que très peu interverti, même dans les plus forts accès." In one severe case Jurine records the respiration as, during the attack 26, and in the interval 23. Raige-Delorme,[2] summing up the knowledge of angina as it was a hundred years ago, says "all observers are agreed that the respiration is not affected . . . or it may be a little quickened." Von Dusch says clearly that the respiration is mechanically free; that if the respiration be quickened it is in fear, or there is a complication. He adds that the diagnosis from cardiac dyspnea, from neurotic panting, and so forth, is not difficult. Watson writes: "Yet the patient is not out of breath." Flint and

[1] Windle, D., *Lancet*, May 13, 1911.

[2] Raige-Delorme, *Dict. de méd.* 22e éd., 1833, vol. iii. p. 142.

Walshe are no less positive ; Walshe, in differing from Stokes (p. 317), says dyspnea is, if present, incidental; that in pure angina respiratory action is " below par." Broadbent [1] says, " Not venturing to make the least movement, and scarcely seeming to breathe." Gallavardin urges the same point. Dr. Morison, describing a particular case, says " the respiration was restrained during the agony, but not otherwise disturbed or difficult." Fränkel says clearly that though angina pectoris and cardiac dyspnea may concur, or in the same patient alternate, yet that these conditions are to be considered severally, though we often fail to do so (" streng von einander zu unterscheiden, wie es vielfach nicht geschehen ist "). Sir Thomas Oliver, noting this peculiarity, asks, " How (in angina pectoris) do we explain the absence of shortness of breath ? " [2] The usual and plausible answer is that the sufferer dare not breathe for dread of instant death, and something must be credited to this deterrent. Josué,[3] in respect both of respiration and pulse, states, " The respiration is free and undisturbed ; auscultation and percussion reveal no modification of the respiratory mechanism, no *fixation of the chest* " (see p. 385). Rosenbach [4] says that not only in pure angina pectoris is there no dyspnea, but even a voluntary refrain of respiration ; " eine willkürliche Hemmung der Athmung, in dem Bestreben möglichst geringe Athmungsexcursionen zu machen " ; as Dr. Morison puts it, " the breathing during the agony is restrained." A case of my own well illustrates the distinction between angina and heart disease in respect of dyspnea.

Male, æt. 26. No rheumatic fever ; denies syphilis, but had been in the army. No very hard labour. For two months—it began suddenly—attacks of severe and piercing pain at the sternum —penetrating, as it grew worse, to the back, and down the left shoulder and arm (in the usual anginous way). During this period of his illness " no physical signs of cardiac disease and no shortness of breath were present " (his own physician's letter). But then severe dyspnea set in suddenly, when on examination a murmur of aortic incompetence was distinctly heard, and the left ventricle was dilating. The case, undoubtedly one of syphilitic suprasigmoid aortitis, pro-

[1] Broadbent, W. H., *Heart Disease*, ed. 1879, p. 298.
[2] Oliver, T., *Lancet*, Sept. 16, 1905.
[3] Josué, *Artériosclérose*, p. 245.
[4] Rosenbach, *Path. u. Ther. d. Herzkr.*, 1899.

ceeded on its usual course from the suprasigmoid area to the valve. Unfortunately after this, the only consultation, I lost sight of the patient.

Notwithstanding, such nowadays is our slovenliness in the use of clinical language, we are ready to give the name Angina pectoris offhand to any grave thoracic crisis with dyspnea. The name "Cardiac asthma" is another instance of such abuse; Asthma of course is not a cardiac affection at all, and is not to be confounded with affairs primarily of the heart or circulation.[1] Asthma is an expiratory hindrance. Yet if, even without pain, the patient fall into an agony of paroxysmal dyspnea, his physician may clutch at the name of "Angina sine dolore," an error of which so great an observer as George Balfour cannot altogether be acquitted (*vide* p. 324). So it comes about that this name is given promiscuously to processes so various as syncopic heart failure, the paroxysmal dyspnea of high pressures, nocturnal cardiac orthopnea, and so forth. A gentleman (æt. 75), in apparent health, while walking up a hill, was pulled up rather suddenly by a gasp and a depressing ache, of no great intensity, about the heart, and for shortness of breath found some difficulty in reaching home. Three physicians, one after another, diagnosed his case as "Angina pectoris"! The attack was in fact, as the subsequent history proved, not one of angina at all, but the breakdown of a long-standing Hyperpiesia; on further examination we discovered abiding high arterial pressure (about 200 mm.), a considerably hypertrophied and dilating heart, and cardiac dyspnea on slight exertion. He lived two years longer, suffered two slight apoplexies, and died of defeated heart, with never a symptom of angina. Such confusion of diagnosis is presumably due, not to ignorance, but to random language, engendered of lax or sophistical arguments. Again, a younger man—a physician, aged about 43—consulted me under the diagnosis of "angina pectoris." His blood pressures ranged very high, and the left heart was big and dilating. There was no symptom nor sign of renal disease. He had been stopped on rising a hill by "a pain at the heart"; but a closer enquiry elicited that what pulled him up was not the ache—for

[1] This distinction I have emphasised elsewhere, as in the *Brit. Med. Journ.*, Oct. 28, 1911.

that he could have proceeded—but an imperious dyspnea. And, on being asked where the pain was, he laid his flat hand under the left nipple and towards the anterior axillary line. There were no radiations, and the ache was not paroxysmal but continuous. The pain was not the distress; the distress was the hard breathing. Unwittingly he also had been for some years a victim of Hyperpiesia, and the strained heart was giving way. In the course of the next few months it yielded more and more; anasarca appeared, arterial pressures began to fall, and, "nearing his end with labouring breath," he too died of cardiac defeat; but he never had angina pectoris.[1] It is because of our respect for our late colleague, Professor Dieulafoy,[2] that I feel compelled to deprecate even in him descriptions of this kind; for the example of so great a clinician leads to the greater misconception. After speaking of the pain of angina as "partie du cœur," he brings in "dyspnea . . . agonizing paroxysmal dyspnœa resembling uræmic dyspnœa," which, he adds, may so dominate a case as to throw the pain into the background, or even to replace it, with or without palpitation. In these words the distinguished Professor masterfully confused two wholly different maladies; namely, the high-pressure dyspnea, so well described by Pal, and angina pectoris. Again, a physician of well-merited distinction, concerning a case of post-diphtheritic myocardial disease in which the patient had begun work too soon, reported that he had an attack in which he became much agitated, and his breathing rapid and panting; he complained also of "distress over the lower part of the præcordium [*sic*]," the pulse weakened and quickened, and physical signs of cardiac dilatation appeared; yet this set of alien symptoms the author discussed unreservedly as an attack of angina pectoris! Again and again by distinguished authors agonising respiratory spasms of high pressure, of uræmia, or indeed of pulmonary embolism or acute œdema, are thus spoken of as angina pectoris. One says, for instance, that "the strain of angina pectoris may arise on the pulmonary side of the circulation," and cites as examples "the not infrequent anginal seizures in mitral

[1] This high-pressure dyspnea is discussed in the chapter on Arteriosclerosis (Vol. I. p. 402).

[2] Dieulafoy, *Path. interne*, ed. 1908.

stenosis," which "may be warded off by hæmoptysis." By the context it would appear that here the author is considering paroxysmal dyspnea (pulmonary embolism ?). In what sense angina may occasionally depend upon mitral stenosis will be discussed on p. 443.

Even the great Stokes, in his unhesitating attribution of angina to failing heart, did not see his way clearly between angina pectoris and the dyspnea which he supposed to be of similar nature. The fact was, as he admitted, in his practice he saw little or nothing of angina pectoris. A like undiscriminating diagnosis we find in one of von Basch's cases:

Male, æt. 54. Angina pectoris extending to the arm, but with great dyspnœa, sense of palpitation, a hurried irregular heart, and death. The attack permitted the estimation of arterial pressures which fell from 160 to 105.

This was a case of angina, no doubt, but of angina complicated with some grave cardiac lesion. Yet this master of his subject allows himself, without qualification, to call this mixed case an "angebildeter Fall" of angina pectoris.

Let us remember then that the distress of angina pectoris is not any paroxysmal thoracic distress, but a peculiar distress; distress of cardiac origin, or of high pressures, whatever its gravity, is of other kinds; it is the horror, not of a crushed thorax, not of the bottomless pit, but of the garotte. In compound cases, one and the same patient may unhappily have an experience of both miseries; sometimes he may be attacked by paroxysmal dyspnea, nocturnal dyspnea, or dyspnea on exertion, at another by angina; sometimes the two conditions may fall upon him together, as asthma may appear during bronchitis, or as cirrhosis of the liver may be associated with delirium tremens. It is for this reason that I have ventured to insist at length upon the postulate that dyspnea, be it never so sudden, so violent, or so suffocative, is no essential symptom of angina pectoris. It is usually the first betrayal of cardiac inadequacy.

Of one manner of termination of angina, namely, by gradual heart failure, I have already spoken; from such phases cardiac and cardio-pulmonary dyspnea can scarcely be absent, and usually have made their own independent way. In a simple case

of angina pectoris, however vehement, the results of physical examination of the heart are as likely to be negative as positive ; in cardiac dyspnea, on the contrary, a negative result of examination of heart and lungs would rarely happen. Paroxysmal "toxic" dyspnea—bulbar dyspnea, if it be fitly so called—is not an agony of pain, but a desperate fight for breath. It is true, as Vulpian, Huchard, Pal and other observers record, that, if now and then in an anginal attack respiration be not arrested, its rate may be increased, even considerably ; but, as these authors have noted, in such cases the respirations are shallow and hasty, with a more or less restrained thorax, and are not, of themselves, a cause of distress, nor indeed of much importunity. As Vulpian (*Clin. Med.*) remarks, if in angina there be any dyspnea, it is not of the cardiac form ; it is a retarded and somewhat spasmodic sighing, like slight asthma. Finally, although in some mixed cases certain remedial means, such for instance as those which reduce arterial pressure, may mitigate both the angina and the dyspnea, as by breaking a vicious circle, yet, broadly speaking, the therapeutical methods which we bring to bear upon cardiac dyspnea differ considerably, even essentially, from those in which we have learned to confide for the relief of pure angina. That Balfour, in his opinions upon angina pectoris, was not always consistent with himself, I have ventured to suspect, as I have questioned his use of the term angina sine dolore (p. 324) ; but the independence of angina both of asthma and of cardiac dyspnea, he never failed to perceive.

However, Erasmus Darwin (Zoonomia) thought angina akin to asthma, and related a case of their coexistence. The suggestion recalls to me a compound case of angina and asthma which I saw many years ago with a physician in Ripon :

Mr. T., a man in later middle life, had been attacked by angina pectoris presenting the usual characters. On listening to the left side of his chest behind, the respiration was audible ; but on transferring my ear to the right mid-lung it was inaudible. I tapped the side anticipating the signs of fluid, but the resonance was normal. Puzzled for the moment, and then diverted by some agitation of the patient, both I and my colleague were surprised to find him (to cut the story short) under a seizure not of angina but of asthma. I think the patient had suffered from asthma in former years, but not for a long time past. It seemed that in the excite-

ment of the consultation the bulb (?), vexed by attacks of angina, lapsed on the line not of angina but of asthma. He had suffered from angina only on walking uphill.

My impression is that the arrest of breathing or the sighing, seen in most cases, is an inhibition analogous to the inhibition of the heart, of which I shall offer some illustrations later. Inhibition of respiration is often seen during a drag on the mesentery in animals (p. 422). All I will add now is that, as in ordinary respiration the cardio-inhibitory centre is more active during expiration and less active during inspiration, in angina we may suppose conversely that an intenser cardio-inhibitory influence may reduce the breathing in the direction of expiration. Irritation of the interior surface of the ventricles, as we know, inhibits the respiration. Of the three neighbours in the bulb, the respiratory centre seems to be the most susceptible, both from above and below (Brodie and Russell). Wenkebach [1] says, "During compression of the vagus the patient holds his breath, generally ("vielmals") in the inspiratory position (so that the cardiogram appears lower), *while there is no diminution in the radial wave*" (italics mine). But these factors, bulbar, respiratory, cardiac, musculo-cutaneous, splanchnic and the rest, are too complex for easy discrimination.

Angor Animi.—The horror—the *angor molestus* of Morgagni—which in its extremer degree is almost peculiar to angina pectoris, is so well known, and so impressive, that I need not dwell upon its character. To return to Seneca's story of his own case [2] (of angina), "The attack is very short, and like a storm. It usually ends within one hour. . . . To have any other malady is only to be sick, to have this is to be dying." Well might his physician call it a *meditatio mortis* [3]—to die piecemeal. Morgagni, in the case of an elderly woman (xxiii. 8), describes it, "*tanta ad cor angustia atque anxietate, ut saepius quamprimum moritura videretur.*" Extensive aortic atheroma was found at the necropsy. It is not a reasonable apprehension, nor a fright or

[1] Wenkebach, *Arch. f. Anat. u. Phys.*, 1908.

[2] See the interesting essay by Pawinski, "Das Leiden Senecas," *Zeitschr. f. klin. Med.* Bd. lxviii. Hte. 1 u. 2.

[3] *Meditatio* here does not mean a "contemplation of death," but an exercising in death; *e.g.* "*In cursuram meditabor me*"; or, as Pliny the Younger uses the word, an exercising or practice for a campaign.

alarm, as at a snake in the grass, but an organic sensation. As even in moderate cases the pain is menacing, and in severe cases atrocious, and as it is natural to attribute the dread to the vehemence of the pain, it seems difficult to realise that the angor [1] and the anguish can be separable, and presumably therefore may own somewhat different origins. Still these two symptoms by no means run parallel in intensity; in one case we may see flashes of pain, so furious and incessant that, as in the terrible case described by Dr. Knott (*loc. cit.*), the sufferer may well fear indeed to be overwhelmed by them, yet he may not be possessed by the spectral horror which, in another case, may dog the mildest series of attacks; indeed this dread often sets its seal upon attacks in which the pain might otherwise have been equivocal. A step farther, and I am a dead man! In the successive seizures of the same patient, it is true, the blend of pain and horror may be fairly uniform; but a strange indescribable fear is no uncommon feature of cases which, in respect of pain, may be no more than angina minor. Indeed, as I have said, it is often by this touch, rather than by any intolerable pain, that in minor warnings the patient is appalled.

> Verum ubi vehementi magis est commota metu mens,
> Consentire animam totam per membra videmus;
> Sudores itaque ei pallorum existere toto
> Corpore, et infringi linguam, vocemque aboriri,
> Caligare oculos, sonare aures, succidere artus.

Shall we guess that Lucretius also, like St. Luke, Dante, and Shakespere, was a physician? [2]

One of my patients under middle age, perhaps 40, not more, a subject of severe syphilitic angina of four years' duration, told me he had experienced the sense of impending death in one attack only—once in many a score—" and on that occasion the pain was

[1] Angor primarily had the meaning of suffocation or strangulation; Cicero carried the word metaphorically into anguish of mind.

[2] We may recall also in this horror these lines from the *Dream of Gerontius*:

> " Down, down for ever I was falling through
> The solid framework of created things,
> And needs must sink and sink
> Into the vast abyss. And, crueller still,
> A fearsome, restless fright begins to fill
> The mansion of my soul."

not much." Thus by a typical case, as this was, we see that the angor is by no means an essential feature.

My own experience suggests that in cases of angina in persons with sound hearts—for instance in the syphilitic, rheumatic, influenzal, or other kinds of aortitis in young, or comparatively young persons—the horror is less manifest and often absent; it seems rather in older persons, with shaky hearts, that the shadow of this passion may even obscure the pain. Indeed many of these sufferers have more dread of the death portent than of the pain, terrible as this may be. A lady who for seven or eight years has consulted me occasionally for typical angina, the attacks being often severe and persistent, has never had any trace of dread. The attacks are frequent, at times when more exertion is demanded of her, almost incessant, yet hitherto her life has been spared; the closest examination reveals no sign of cardiac disease, nor usually of any very excessive arterial stress, though she has occasional periods of high pressures. So far, the absence of dread has seemed to be a favourable prognostic. Prolonged rest for a day or two puts the symptoms into abeyance, but as she finds inaction very irksome, she breaks through our restrictions; now the patient who has once felt himself hanging over the mouth of the pit will submit to any advice. We shall see indeed that, as in another patient I shall describe, in some rare cases the dread alone may fill the bitter cup.

It is remarkable that this peculiar awe, as distinguished from reasonable apprehension or awareness of the menace of death, this organic sensation, is rarely met with in heart diseases. Even Stokes-Adams' disease—if I may rely upon the symptoms of some exemplary cases of my own—is not, or not usually, attended with this "Todesgefühl." One very intelligent patient, who suffered for a long period from the syncopic and convulsive phases of this disease, told me that the "going off" was but "a dreamy sensation, not in itself unpleasant."

The absence of the sense of impending death, intimate symptom as it is, is then consistent with a diagnosis of angina pectoris; this is proved over and over again, both by its inconstancy in cases of all degrees of severity and, if the diagnosis in such cases be questioned, by its presence in some attacks of the same patient, and its absence in others. In this statement Sir William

Osler concurs.[1] Conversely, Heitz (p. 194) and others note its occurrence in chronic aortitis, in distinction, as they put it, from "coronary angina"! So inconsistent are the coronarians. That in the several attacks of the same patient the proportion of the symptom to the pain may be far from constancy is a point to which I shall return in the interpretation of the disease. As a symptom then the angor animi is frequent, characteristic, and dramatic; but far from universal, and cannot be accepted, as many would have us accept it, as a warrant.

That this awe may depend upon a consciousness of forced inhibition of the heart (*vide* p. 466) presents itself to the mind, for it is met with in some cases of interference with the vagi, such as the pressure of a tumour (Skoda's well-known case), and in a once well-known example quoted in Müller's *Physiology*. In certain old notes on angina pectoris, notes made when I was preparing for my final M.B. examination at Cambridge, I find the following extract from the edition of 1841 (iii.):— "The heart's movements were occasionally checked for intervals of four or six beats, when sensations of fearful anxiety were felt" (with other symptoms). ". . . After death an enlarged bronchial gland was found pressing upon the great cardiac nerve." To like effect Dr. Mott[2] relates, of a highly intelligent man afflicted with a cerebral tumour, that suddenly a sense of fear would possess him, with pallor, momentary arrest, disturbance or weakness of the heart, perturbed respiration, trembling and weakness of limbs, a feeling of anxiety, suffocation, and vague unpleasant sensations in the breast; a fear which came and went with the fluctuation of the other organic disturbances. I am disposed therefore to attribute this symptom to some echo in consciousness of a vagus crisis. Conversely, it would seem that fear may strike down the vagus path with fatal result. Fretfulness, anxiety, and forebodings are of course common symptoms of all kinds of heart affections; and we are accustomed, no doubt, to speak of vagus inhibition—as perhaps in certain intermittences of the heart—as common enough, and unaccompanied by no queer sensations. Recent researches however

[1] Osler, Sir W., *Angina Pectoris*, p. 51.
[2] Mott, F. W., *Brit. Med. Journ.*, April 4, 1908.

are making it doubtful if irregularities of the heart are to be referred to the vagus so readily as has been our wont.

Angina sine Dolore.—In his now classical letter to Heberden, The " Unknown " stated that, amid major attacks, occasionally he suffered from transient seizures (without pain) in which, as it seemed, " there was a general suspension of the inner operations of nature for three or four seconds." On this report, Heberden remarks that in some 50 cases, which up to that date he had observed, he did not remember ever to have heard this mode of angina mentioned. Watson,[1] on Quain's authority, says " the inexpressible sense of dying is sometimes the only symptom of the disease." In Miss Mamie Dickens' recollections of her father, it appears that Dickens, for some time before his death, had been subject to attacks of vague but overpowering dread, and in one of these attacks he died. Charcot's single attack, in which he died, may have been of this form.[2] What can that sinister touch be which, until the final stroke, does not even make the heart falter ? To identify this spectral visitation with angina does indeed sound like a contradiction in terms, as if one should say angina sine angina ; and had I not witnessed an exemplary case of this extremely rare variety of the disease, I might have continued in some scepticism concerning it. The title should indeed be used with the utmost discrimination ; we have to regret that into this accommodating pigeon-hole are stowed all sorts of obscure catastrophes or sudden death. For authors to whom any release from the discipline of facts is welcome, the name is happy enough, for it will fit almost any painless but startling thoracic distress. Accordingly it is made to fit events so alien one to another as syncope, bradycardia, paroxysmal dyspnea, cardiac or toxic, any fatal stoppage of the heart, acute pulmonary œdema ; or, on the other hand, the comparatively harmless " vaso-vagal " storms. On the use of this name Gairdner himself wrote, in consent with me,that its indefiniteness made its application too often ambiguous or otiose. I quote the pertinent portion of his letter, dated June 15, 1894 :

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[1] Watson's *Lectures*, vol. ii. p. 285, ed. 1857.
[2] See Parkes Weber, *Medical Magazine*, Nov. 23, 1893.

" Angina sine dolore " is perhaps entitled to be called an " enfant gâté," but if so, has not been coddled into notoriety by anything done for it, or to it, by its unworthy parent, but rather by its having somehow " caught on " in the shape of reference by outsiders to whom it has seemed to convey something that was within the range of their experience. What I believe to be true about it has been briefly and modestly set forth in a few words in my article in *Reynolds*. I have always marvelled a little at this phrase coming up again so much as it has, when better things have been left in the cold shade of neglect, showing once more how much lies in a name.

May I add it is one more illustration of the truth that we invent catchwords at our peril; they are handy to catch popular impressions of things we have never thought out and, with the new resource of a name, need not think out. Our brilliant French colleagues have helped us to too many of these dialectical counters. To quote examples of the ambiguous use of the name Angina sine dolore is no very gracious task; but may I not reason in this matter with George Balfour, a memory too great to be touched by my cavilling? When Balfour [1] writes " termination suddenly in asystole, death from angina sine dolore," may we not ask, Why angina? Cannot a man die suddenly of heart disease without angina? And in a following, and surely inconsistent, sentence, he writes, " Cardiac asthma is another variety of angina sine dolore," the very error we have just been deprecating; for the distinguished author himself had written on angina pectoris (p. 315)—" There is nothing pulmonary in this seizure; the air goes less freely into the lungs *if the patient has the courage to breathe* "; and he proceeds, " it has no connection with spasmodic asthma, *nor with ordinary cardiac breathlessness* " (italics mine). Let us take again the following case, published by the friend lamented by so many of us, Dreschfeld of Manchester, and quoted by later writers as one of angina sine dolore, yet surely in no sense a case of angina pectoris at all? Briefly thus:

Female, æt. 49, one night suddenly was seized with severe dyspnœa, but not with pain. Seven days later a repetition of the seizure with fatal issue. At the necropsy, left coronary artery occluded by an atheromatous plate; the apex of the heart the seat of fibrotic degeneration. Blood pressures not recorded.

[1] Balfour, Geo., *Diseases of the Heart*, 3rd ed., Sect. " Heart and Aorta."

All we know then of this case is that the heart was gravely diseased, that of this disease the patient died suddenly, and that the eminent symptom was dyspnea. But we have seen that dyspnea is not a symptom of angina, dolorous or indolorous. However, it is true that angina sine dolore, if extremely rare, does occur; I will now record a genuine case of it, the one well-marked consistent case which has occurred in my own experience; fortunately I witnessed a characteristic attack.

A gentleman of some 70 years of age was on his way from the North to visit me in Cambridge, when his illness so increased upon him that he was laid up at an hotel in London, whither I went to him in haste. I found the patient, a pallid, worn-looking, but not emaciated man, in his dressing-gown, seated in an elbow-chair in his bedroom, and surrounded by the aids and restoratives of the sickroom. The radial artery was large, thick, and tortuous, and the pulse thrusting; the heart was large and labouring, with evidence of atheroma in the aortic area. While I was examining him he blanched, either with fear or in some organic vascular tide, and in a hollow voice whispered, "It is coming, it will kill me." For a few minutes he sat thus stricken into stillness; until his countenance began to open, he drew breath, and moved on his chair. During this interval, some three minutes perhaps, while his servant, familiar with these attacks, applied some stimulants, he did not disengage my finger from his pulse, which had not faltered, nor betrayed any vacillation, but became transiently a little slower and more laboured (longer diastole and larger output?). In these attacks of sudden dread, terrible as they were, and he had had not a few of them, there had been no pain whatever, not, I believe, in any one of them. He was fain to wish for any alternative suffering to compete as it were with this death-like touch. "A faint cold fear thrills through his veins, that almost freezes up the heat of life." In one of these attacks, a few weeks later, he did die, suddenly.

Neuburger (*loc. cit.*) reports that in exceptional cases an exhausted sensation, or intolerable depression in the region of the stomach ("Magengegend"), especially in the "epigastric" cases, and after a full meal, may take the place of the pain. Mrs. M., a patient sent to me by Dr. Forrest of Terrington, complained more of the "sense of fearful faintness and of

death," than of the characteristic pain. She was subject to occasional nocturnal attacks, without the pain, which may have been wholly of this kind. They seemed to begin in a dream of annihilation.

From my experience, such as it is, and from the records of a few similar cases, I am of Gairdner's opinion that a category of Angina sine dolore should be recognised; but that it must contain cases only in which such paroxysmal crises, if devoid of pain, are marked nevertheless with the seal of the death terror, and indeed chiefly consist in this; and are not to be confounded with startling symptoms of cardiac or cardio-pulmonary disorder. Moreover the crises must be paroxysmal, and disposed to recurrence. Of course a first attack may be fatal; if so, the diagnosis could not be decisive, not even, in the present state of our knowledge, with a necropsy. Even if the patient should have time and breath to describe his sensations, it would rarely be possible at so brief and critical a moment to discriminate between it and the alarm which may accompany an ordinary cardiac crisis. But of course in mixed cases, as with anginal pain so with angina sine dolore, there may be a complication of cardio-pulmonary, renal, or other concurrent malady.

Some years ago I saw an Essex squire with Dr. Prince, then of Buntingford, a very old man, in whose case attacks of angina sine dolore were now and then intercalated between the ordinary seizures. Dr. Prince's description was quite characteristic, but I myself did not witness one of these. Sir John Broadbent has kindly reminded me of the following passage in his father's book on *The Pulse* (p. 178): "The patient would be seized in the street with sudden faintness and deadly apprehension. The attacks ceased with the disappearance of the high tension from the pulse." In him I suppose the suprasigmoid aorta to have been in a morbidly sensitive state, and the already excessive stimulus to the vagus intensified, so that the automatic regulation of cardiac energy and aortic pressure, which is continuously at work in all of us, in him became so unbalanced as to enter into consciousness. Schmoll (*loc. cit.*) describes a case of angor animi with slight numbness and paresis of the left arm, but no pain.

To sum up, I repeat that, dramatic and ghastly as is this *metus deliquii*, we must not take it as a criterion, nor declare that without it there is no angina. Merklen agrees it is not "pathognomonic," as he considers "retrosternal constrictive pain to be, especially if associated with the slightest numbness of the left arm." And as we have seen, it may accompany vagus irritation under other conditions. This Sir William Osler[1] has made quite clear, and by many cases proved, that it may be absent, as in epilepsy aura, or convulsion, may be absent. But I do not agree that the angor animi is called forth by the severity of the pain. We have seen that anginal dread may occur without pain; and although it is true that a bad case is prone to be bad all round, still I would urge that there is many a case, especially of acute aortitis in young persons, in which the pain is extreme, and yet without angor.[2] If I may venture to repeat my own notion on this point, it is that the dread is more frequent in degenerative cases; and is often absent in the acuter angina, such as the syphilitic, the influenzal, or the rheumatic, of younger persons.

Once more; we cannot, in cases of sudden death by the heart, predicate angina as the cause without either some report of the peculiar pain or of the peculiar organic dread; not indeed even if the patient had been a subject of angina.

Immobility, in a wider range than of the respiratory system, is the ordinary condition of an anginal attack, and the contrast of this with neurotic agitations has often been pointed out. Heberden graphically describes how these patients, walking hastily, or against a cold or contrary wind, or up a hill, "simul ac pedem figunt et quieti sese dant; subito plerumque omne incommodum evanescit." "The pain," said one of them, "compelled me to sit down." Fothergill, and Gruner,[3] concur in this description. Forbes also graphically describes the sudden pause, the extension of a hand to some support—"or it may be the patient imperceptibly sinks down on a chair or bank, as if unable to stand, yet afraid of the movements necessary to seat him."

[1] *Loc. cit. Ang. Pect.* p. 51.

[2] See Sir W. Gowers, *Brit. Med. Journ.*, July 21, 1906; and Dr. Mildred Burgess, *Lancet*, Nov. 12, 1908. But in these passages the authors scarcely distinguish between an organic passion and a reasonable fear of death.

[3] Gruner, *Spicilegium ad Anginam Pectoris*, Jenae, 1787.

And yet in this he saw nothing to separate such a summons from the mere shudders of vasomotor oscillations!

But rest is no warrant against an attack. Parry,[1] and Gruner—and of course many later writers—describe attacks arousing the patient even from sleep; a point of diagnosis, when it so happens, from spurious anginas. Of one case of angina, due to an aneurysm just above the heart, Morgagni wrote, "Noctu pectoris angore molestissimo, cum suffocationis metu, corripitur." To explain this, as Obrastzow says, we need not speculate with Romberg and Ortner on diminished circulation in the heart in sleep, when as the rate is then slower and diastole longer, less nutrition is needed; we may be content with a thrust-up diaphragm or too good a supper. The slower and shallower nocturnal respiration in sleep which favours cardiac dyspnea probably has no bearing on angina.

Although for the most part, nearly always, the patient is, as it were, frozen for the moment into stillness, yet it is not true that agitation never accompanies attacks of true angina. In some such stories the diagnosis may have been erroneous, but Dr. Mackenzie,[2] who is not likely to have erred in this respect, speaks of such a patient "rolling on the floor in an extremity of agony." In the anginous cases, which I shall describe later, of acute plug of a coronary artery restlessness is often terrible, but the pain may be incessant for days. The "Unknown" said if he did not "indulge" the pain he could walk it off; and one of Sir William Osler's patients, by a strong effort of the will, could walk the pain off; yet this was possible only in moderate attacks. Probably in this, as in many other such instances, as the muscular and cutaneous circulations open out, the intra-aortic pressures give way, and thus the tension is moderated. Mr. Sumner it was, I think, who found that he could shorten the attack by walking the floor, "but it increased the agony at first." Parry quotes the case of a lady of great force of character, who could compel herself to walk on, when in five to ten minutes the pain would be dispelled. My own experience has been that in single and transient attacks, however severe, stillness has been invariable, but that pacing,

[1] Enquiry into symptoms and causes of the Syncope anginosa, C. H. Parry, M.D., 1799. (In a note the author corrects *anginosa*, which he says is not strict Latin, to *angens*.)

[2] Mackenzie, Jas., *Heart Diseases*, p. 45.

rocking, or sometimes agitated movements have accompanied the more protracted assaults. Jendrassik says he has seen no more movement than an instinctive desire to get the hands forward, or upward and forward; one of his patients begged the bystanders to hold his arms up. Or we may see a movement, even a large movement, made to secure the position of least agony, often a strange posture. In mock angina the agitation is primarily psychic, and often attended with whimpering and choking, plaints which before angina pectoris are silenced.

Pulse.—The symptom next to be considered, namely the pulse, requires a close scrutiny. My own experience, which fortunately has included the witness of many seizures, and in a fair number of sufferers, is that in the large majority of them the pulse was unaffected; and that abnormal characters, if any, belonged to some independent disorder. I have said that, amid the agitation of patient and attendants, the heart, assumed to be the protagonist in the conflict, often seems to be the one impassive actor. Here again we find how much more definitely the early observers of angina pectoris wrote concerning its clinical features than later writers have done. By them this imperturbable attitude of the heart was taught distinctly; though it is true that von Leyden, Osler, James Mackenzie, Morison, and other recent authors give the same clear testimony. Morgagni writes (*Ep.* xxiii.): "Huic pulsus nunquam intermittentes." Heberden noted that often neither pulse nor heart were disturbed, even at the height of a paroxysm. His words are: "The pulse is at least sometimes not disturbed by this pain, consequently the heart is not affected by it, which I have had the opportunity of knowing by feeling the pulse during the paroxysm." Parry says there is never any palpitation; and the pulse is usually little altered, so little that one would not suppose the heart to be affected. He adds that assertions about the pulse are too often mere hearsay, not of eye-witness. During the attack however, as the face pales, the artery often constricts. Burns made too much of such pulse changes as may appear in dilapidated cases, and the name "Syncope anginosa" probably beset him; but he adds nevertheless (p. 144) that "the pulse is not so much affected as one would expect." Jurine[1] records the pulse of a patient during

[1] Jurine, *Mém. sur l'Angine de poitrine*, Paris, 1815.

the attack as 86, and in the interval 82. He had never had his finger on the pulse in the articulo mortis of angina; but otherwise he had never detected either palpitation or intermittence: often it is "serré" and a little accelerated; the face often pale, but not always. "How," he reflects, "if angina be due to coronary disease, can the heart go on steadily in this way, and then drop out suddenly?" Desportes asks the same question. Van Dusch[1] says the heart's action during an attack seems to be little altered, unless the seizure be fatal; occasionally, he adds, we find some enfeeblement of the heart's action, but very rarely palpitation; though this he noted in a case of aortic insufficiency in which "the action of the 'colossal heart' was very energetic." Balfour bears the same witness. Latham, of a severe paroxysm of angina in a man æt. 64, reports: "Auscultation found his heart beating with a perfect rhythm, and neither with excess nor defect of impulse, and its sounds were natural." Walshe says decisively: "In the very extremity of pain the pulse may be perfectly regular, and barely exceed by half a dozen beats per minute the rate normal to the individual." Eulenburg and Guttmann,[2] who carefully noted this point, found the action of the heart and pulse, unless for an occasional slight "Irregularität," to be normal all through the attacks. They felt assured that the explanation of the attacks could not lie in alteration of the heart's action. Fränkel writes: "The pulse is either not changed, or deviates (*abweicht*) but little from the norm." Edgren[3] reported that during the attacks the "definite objective changes in the heart's action, pressure-wave, or frequency, are as a rule not important; *on auscultation the heart seems quite quiet, and just as in the intervals of pain*" (italics mine). This is his conclusion, "having examined many cases curiously." In one case, in which death in an attack was sudden, Edgren had several times (*mehrere Male*) in previous seizures found the heart's action quite tranquil (*vollkommen ruhig*). In two other cases, which ended in status anginosus and death,

[1] Van Dusch, *Lehrbuch d. Herzkrhtn.*, 1868, p. 33.

[2] Eulenburg and Guttmann, *Die Pathologie d. Sympatheticus*, Berlin, 1873.

[3] Edgren, *Die Arteriosklerose*, Leipzig, 1898, note, p. 227. In another place he says, "Bei den Anfällen . . . könnten keine bemerkenswerthen Veränderungen des Herzthätigkeit oder des Pulses nachgewiesen werden."

the heart, except for a slight occasional irregularity, was, until the final stage, for the whole time almost unaltered. Von Leyden [1] testified emphatically that by auscultation there is plenty of evidence that during attacks changes in the sounds or pulsations are rarely observed. Dieulafoy [2] wrote, "During the attack . . . auscultation reveals no abnormal sound; the heart beats are normal or retarded ("ralentis"); *if the angina is associated with cardio-aortic lesions* they may become irregular; but in some cases they are accelerated" (italics mine). In a case on my notes, a certain patient, who had had a few attacks of angina, but was able to do a good deal at times, to take long walks, and go to business, was under medical care for bronchitis, and his own doctor was feeling his pulse; it was steady and regular. But at an instant he cried out, and then whispered, "I am losing myself"; the pulse without a falter had suddenly stopped in death. Till then the heart had presented no abnormal signs. Pawinski, in the interesting paper on basic pericarditis as a cause of angina pectoris to which I have made repeated reference, after noting the disturbance and embarrassment of the pulse in more extensive pericarditis, says of the basal form that, severe as the angina may be, the pulse may be a little retarded, or normal; *unless, with extension of the inflammation, dyspnœa set in with other symptoms of heart affection* (italics mine). In another paragraph he says that a pulse, previously of high tension, as the heart becomes involved may soften and quicken; and conversely, that not a few cases are on record in which the heart, irregular between the attacks, became during an attack normally rhythmic. Dr. Mackenzie [3] writes: "In the vast majority of cases I could detect no change in the heart or arteries, and there never was the slightest enlargement of the heart coming on during an attack." We do not realise what these large admissions signify. In another place he says, it is true, in the small minority "the pulse may become small, soft, and scarcely perceptible, from weakness of the heart" (or from vagus inhibition ?). Sir James Goodhart writes that, as a rule, the pulse is

[1] Von Leyden, *Zeitschr. f. klin. Med.*, 1884.
[2] Dieulafoy, *Path. interne*, 1908, vol. i. p. 1045.
[3] Mackenzie, Jas., *Diseases of Heart*, p. 47.

wholly unaffected, either in rate, rhythm, or tension; and this he states, "having had my finger on it through the spasm many a time. And yet," he exclaims, "we call it heart disease! Why, for the life of me, I cannot imagine. These are not the symptoms of heart disease. The heart has too much grit in it to tumble down dead, except under well-known and definite conditions." [1] In one case he carefully watched a series of paroxysms lasting half an hour, and reports, "his pulse never altered a bit." [2] Of another case he says "the pulse neither altered nor faltered in any way. From the pulse (in many cases) you can get no indication of any value of the gravity of the attack through which the patient is passing." Dr. Verdon (*loc. cit.*) says that of 62 observations during angina he found in 30 no change of pulse, radial or jugular; in others unimportant changes. Moreover he found *all five* myocardial qualities intact. Josué [3] reports: "The pulse is normal, regular, unchanged, though sometimes it may slow down, or hasten a little (*légèrement*). One finds indeed on auscultation the same conditions during the attacks as during the intervals; there is no change in any particular." Now let us ponder over such statements as these by skilful and experienced clinical observers: testimony which could be collected in greater abundance.

When however the heart's rhythm does alter, the alterations are broadly and naturally divisible into the early and the late changes. Early changes, if perceptible, may consist in retardation and sometimes an unevenness in rhythm, even to intermittence; the later may be acceleration and arrhythmia. Although I have spoken of the heart as often or usually impassive, yet not uncommonly by the sensitive finger there is to be noted in one or more beats a deliberation, an impression of delay which may or may not become still more manifest to touch. Many observers who have declared the rule in ordinary cases to be that the heart is little, if at all, disturbed, make this exception of a slight occasional lag. It reminds one of the momentary self-collection of a horse before he leaps (p. 469) But this transient hesitation is to be distinguished from any element

[1] Goodhart, Jas., *Lancet*, July 1, 1905.
[2] Goodhart, Jas., *Clin. Journ.*, April 4, 1894.
[3] Josué, *Artériosclérose*, p. 245.

of heart block; if degenerative disease about the root of the aorta invade also, as well it may, the tract of Gaskell and Kent, the case is complicated by this lesion; it is no longer a case of mere angina. Thus in one of Osler's list (Case XXIII.) the rate fell from 96 to 42; in one of Verdon's to 40. It is possible that a very angry vagus might make such an effect; but it is in the cases complicated with dilapidated heart and gradual dissolution that acceleration or arrhythmia may set in; albeit it is during this hesitant phase of comparative stability that the heart is prone to trip over. Osler quotes a case of Thayer's in which this phase was very brief; the pulse stopped almost at once; and in others of the few cases where the pulse happened to be in touch at the moment of sudden death, the arrest seems to have been nearly or quite instantaneous. On the approach of death in angina many authors have taken note of swerves of rhythm or falls of pressure.

If in some cases the arterial pressures are excessive, in the majority the pressures are normal; or at any rate not in excess of the standard for elderly persons (Osler, Morison, Gibson, Allbutt, and others). In the angina of younger persons—as in influenzal, rheumatic, or syphilitic aortitis—the arterial tree and the pressures are indifferent, except in so far as pressure may be driven up temporarily by the attack. Dr. Morison,[1] of a case which was long under his observation, says, "The pulse was not quickened, but during the pain it occasionally intermitted, as we know experimentally often happens when the vagus is faradised; after the attack passed off the pulse was smaller and more rapid than during the persistence of the pain."

When the mortal issue is deferred till the later stages of illness, and a period of acceleration and arrhythmia comes about, the failure of the heart is more gradual and intrinsic; but the two modes of death may cross each other, the period of exhaustion being suddenly and mercifully cut short. Dr. Uhtoff of Brighton kindly sent me the notes of such a case in a lady, æt. 61, subject to severe attacks of angina. In her last seizure the pulse, which had been feeble and irregular, was improving under treatment, becoming steady and even regular, and she was talking quietly when, his hand still on the pulse, without any preliminary stagger

[1] Morison, A., *Cardiac Pain*, p. 287.

or falter the beat suddenly stopped. At the same moment pallor overspread the face, the gaze fixed; then came lividity, a convulsive movement, failure of breath, and the end. Under rise of pressures the pulse may slow down; but a slowing due to the vagus signifies, not a rise, but rather a fall of pressure. Again, in Sir William Osler's treatise on Angina, I may refer to his case of status anginosus in which, after some fourteen days of agony, the pulse rose to 115, and began to fail in volume, while the sounds of heart became enfeebled, and the ingravescent dilatation became manifest by the usual symptoms and physical signs; but this patient also in his last attack died suddenly. The experience of authors who assert that in ordinary cases of angina the heart is agitated—as, for instance, when so able a physician as von Basch speaks of angina pectoris as "that turbulent disorder of the heart" —must have been peculiar; or they have confused simple angina with angina plus heart affection; or described what in their opinion ought to have been rather than what was.

Dr. Mackenzie, as I have said in another paragraph (*vide* p. 471), has laid emphasis on the *pulsus alternans* as an occasional phenomenon of angina. Dr. Davenport Windle also has published,[1] with analyses of the pulse and respiration curves, some interesting cases of angina with pulsus alternans; but his cases were so variously complicated with cardiac and other diseases as to be of little service in this narrower enquiry. Although the symptom has no direct connection with the angina,[2] and although in cases of high pressure it may be a sign of aortic tension rather than of intrinsic default of the heart, yet it is so significant of the perilous susceptibility of a wavering heart to interference, that I shall return to the subject under the head of Inhibition (p. 472). (See also remarks, Vol. I. p. 401, in essay on Arteriosclerosis.) I have observed this form of pulse very plainly in the stage of cardiac hypertrophy, in nephritic hearts for instance, when indeed, under the long persistence of high pressure, the ventricle is beginning to "give." The pulsus alternans is not of course necessarily, nor associated often, with angina; Dr. Mackenzie[3] records a case

[1] Windle, D., *Lancet*, May 13, 1911.

[2] Since this was written Wenkebach has since reviewed this symptom in his work quoted p. 476 *n*.

[3] Mackenzie, Jas., *Brit. Med. Journ.*, Oct. 20, 1906.

of angina with pulsus alternans in which the maximum systolic pressures were 190 mm.: now the angina (which recurred from time to time on exertion) was always promptly relieved by amyl nitrite, yet this relief was unattended by any alteration of the pulse; indeed the alternating rhythm was more marked in the intervals of the angina, when the pressure had risen to 200. A certain anginous patient of my own, æt. 45, found no relief from any form of nitrite; with other signs of weak and dilated heart he had pulsus alternans; at a cuff pressure of 140 mm. only about 50 per cent of the beats came through.

Of *volume and tone the changes are inconstant*; sometimes the radial artery is constricted—"serré" as say Desportes and Jurine—scarcely however so generally tight as Sir Lauder Brunton suggests; in elderly persons, for instance, we meet, even more frequently, with the large leathery vessel characteristic of their time of life. Brunton's sphygmogram, published in 1897, which was said to represent such a constriction during an attack, has obtained a great vogue, because it led the author to prescribe nitrites with the success which has become memorable. Yet, in my opinion, this very success has blinded us to other facts which suggest that this sphygmogram was a more eloquent testimony to the diligence of the experimenter than to the capacity of the sphygmograph. The instrument is incapable of analysing a pulse such as that recorded; evidently there is no articulation in the curve, and such superficial form as it has is susceptible of more than one interpretation. We have a far better testimony in his remark that under his finger the pulse felt unmistakably tight. As a basis for hypotheses, such as the vasomotor determination of angina, it is, as probably Sir Lauder himself would admit,[1] almost worthless; probably he has relinquished his maxims—no high pressures, no fear of angina. I have found the radial in angina now constricted, now dilated; and cases are recorded in which the vessel was constricted in one arm, but of full diameter in the other. Of two such cases of my own one was clearly a "vaso-vagal" case, and in the other atheroma had

[1] The same criticism is no less true of Sansom's series of sphygmograms in Angina Pectoris (*Diog. of Dis. of Heart*, 1892, pp. 429-30). The curves in attacks Fig. 104 1 and 2 are quite undefined, and this is even still more true of Fig. 105 1 and 2; the arteries are too contracted to show details; the second may show a rise of pressure. No. 1 (aortic insufficiency) might be quite a healthy curve.

probably encroached upon the mouth of the subclavian. Von Leyden says (*loc. cit.*) the artery in the attack may become smaller or larger. Sir James Goodhart writes:[1] "That high tension is the sole cause, or even the usual cause, of some of the most typical cases, I am quite certain is not the case. . . . I have had a finger on the radial in angina pectoris from the beginning to the end . . . and I am certain there has been no excess of tension of any kind." He adds that Hilton Fagge was doubtful as to a high tension in all cases; and both authors have argued that, if decrepit, the heart must be incapable of maintaining it. Arterial pressures may rise, and vessels may constrict, under any painful impressions. However Sir William Osler[2] remarks, of a case in his consulting-room, that the pulse rose to 90, and became smaller and harder"; though he was "surprised the pulse changed so little;" afterwards, "as the attack subsided, the pulse became softer and fuller, and of decidedly lower tension." Sir Richard Douglas Powell relates a case in which during the attack the pulse was thready, very small, scarcely perceptible, but very hard (see Sansom p. 341). In another case he speaks of the pulse as small and thready; and in a third "An attack of pain came on attended with quickened action of the heart, and diminution in size and increase in tightness of the pulse; the diminution in size proceeding almost to extinction, a mere tightened thread being felt under the finger. Dr. Morison observes that a dwindling of the artery might signify a syncopic decline rather than an exacerbation of tone; in one case (*loc. cit.*) he reports of the radial: "Its palpability varied: it was at times large and more easily felt, and again smaller and less palpable." This fall may happen no doubt, but, as we have seen by general testimony, it is not the rule. A medical man of large experience, who consulted me for angina, found that his blood pressure, which usually ranged from 125-135 (his age was then 67), when an attack came on, "jumped to 150 or more." Here however we have the anxiety of a medical man observing himself. Dr. Hunter of Glasgow has narrated a case[3] in which blood pressures were taken during severe

[1] Goodhart, *Clin. Journ.*, April 4, 1894.
[2] Osler, *loc. cit.* p. 79.
[3] Hunter, W. K., *Brit. Med. Journ.*, Oct. 16, 1909.

attacks. In tranquillity the pressures ranged about 145-150; during a paroxysm pressure would rise even to 240; one minute after a dose of nitrite of amyl it would fall to about 150, and then rise again to about 190, until the attack had ceased, when it would have fallen again to 150. The attacks would last from a quarter of an hour to an hour, with temporary reliefs by the amyl. At the onset of an attack the patient "felt his heart (which was large with aortic regurgitation) becoming stronger and stronger." Dr. Hunter thought that the rise of pressure was not secondary to the pain, but the initial factor in the attacks.

But these are only systolic pressures; the amplitude, which I have measured in the acuter stages of anginal aortitis—syphilitic or other—may be enlarged; and Dr. Mackenzie has proved instrumentally, what my finger had perceived, that in the attack there need not be, and often is not, any rise of pressure. The coronary hypothesis implies a fall of pressure. Merklen (*loc. cit.*), in some carefully observed cases of angina, found the blood pressure never exceeded 125. Speaking generally, in the syphilitic cases, and they are many, the arterial pressures are not increased. Sir Thomas Fraser has remarked:[1] "Sometimes there were angina pains, with no tenseness of vessels, a condition I am unable to explain." The explanation surely is that at the focus of the disease the vessel is so sore that pressures not præternatural suffice to exasperate it. Schabert,[2] in a series of careful measurements in many kinds of disease, found in two cases of angina that during the attacks the systolic blood pressure fell (vagus effect?). Gibson also recorded in a case of angina a fall of pressure during the seizure[3] (under vagus influence?). In another curious case[4] which seemed to me however to be more—probably a case of neuritis, or pressure from growth, than of angina?—he found the pulse during the attacks of pain to contract, but on administration of amyl nitrite to expand by bounds. I observed in a certain high-pressure case that attacks might come on during remissions of the pressure.

[1] Fraser, Sir T., Cardiac Tonics, *Edin. Med. Chir. Soc.*, Feb. 6, 1895.

[2] Schabert, *Petersb. med. Wochenschr.*, 1911, No. 4; quoted *Deutsche med. Wochenschr.*, March 2, 1911.

[3] Gibson, G. A., *Edinburgh Hospital Reports*, 1895, vol. iii. p. 250.

[4] Gibson, G. A., *Brain*, 1905.

It seems then that the blood pressure in angina is inconstant; it may be excessive, normal, or even low; the psychical element, as in the case in which I found the pulse amplitude increased, must affect the pressure, in some cases considerably. In accordance with these observations on the heart, is the cognate testimony that in mere angina the brain remains clear—"even," says one physician, "when death is imminent."

Or a change of tone may be but local. Heberden reports a case of asphyxia of the left forearm and hand, in which during the attack the extremity would suddenly become cold. Trousseau also reports a case where extreme pallor was followed by a pronounced violet and bluish coloration of the arm and hand, the natural colour being restored as soon as the pain ceased (*vide* p. 298). Balfour records of a lady that her attacks were always preceded by pallor of the face and fingers; and Bristowe speaks of the affected limb becoming pale and cold. It seems then that in angina pectoris the vasomotor conditions, both general and local, are inconstant. It is probable that by the disease of the aorta its sensitive endowments, those which regulate blood pressures, are disordered — exalted or impaired; and, during or near an attack, pain, dread, or distress make all measurements of blood pressures untrustworthy. As regards atheroma of the coronary arteries, I may remark here that their decay is more frequent and extreme in decrescent than in high-pressure arteriosclerosis; if of coronary origin, angina should therefore be a low-pressure disease.

Sweating may be a feature of any conflict, of any agony; it is a curious character of angina pectoris, as it is of uræmic or hyperpietic paroxysmal dyspnœa; and likewise is variable, some patients keeping a dry skin. In one of Dr. Morison's [1] cases the patient felt cold in the attack, and did not perspire during or after it. Possibly in some there may be areas of inhibited sweat, to be detected by Dr. Purves Stewart's black-powder test. Osler writes that occasionally sweating is absent; I can say that it may be so slight as not to attract attention. But its occurrence seems to stand in some more intimate relation to the symptoms than as a function of effort or prolonged distress. In most cases indeed the effort is one of endurance and fortitude

[1] Morison, A., *Cardiac Pain*, p. 32.

rather than of muscular conflict; moreover the sweat may break out before the torture is applied, even before it is imminent. Such a case I saw, at the R.A.M.C. examinations in February 1908, in a subject of syphilitic arteriosclerosis and aortitis with angina pectoris. The attacks were severe and not infrequent; the pain travelled in the usual course, according to type, but stopped precisely at the pisiform bone. Now in this man a profuse sweat always preceded the attacks by half an hour. Thus the patient could foresee, not only the return, but also the probable severity of a seizure; for before the severer seizures "sweat would break out all over him, and drop from his whole body; then the angina went into his breast like a knife." Morgagni, in one of his cases ("insultus vehementiores, angore molestissimo cum suffocationis metu"), mentions "sudor copiosissimus" as remarkable. Dr. Mott, in his recent Lectures, refers the cold sweat to an excitation of the sympathetic, with contraction of the involuntary fibres of the sweat glands; some recent observations have suggested that the vagus also may have some influence upon the secretion of sweat. In this connection the pupils and palpebral fissures should be noted. A critical erythema of the face, neck, and upper chest, with a sensation of warmth, is said by some, I think by Eulenberg, to mark now and then the abatement of the attack, especially in women. Sometimes the colour appears in small red patches, like measles.[1]

A flow of saliva has often been recorded during an attack, or after it. Dr. Mackenzie explains this as a cardiac reflex acting through the vagus on the lingual nerve; but I shall return to this symptom presently. The difficulty in swallowing, observed in some cases, may own the same cause. This symptom likewise, as other secretory, paræsthetic, or vasomotor conditions, may forerun the attack.

Fever is present in the acuter modes of aortitis, as in the rheumatic. Often of course it escapes attention, but, in cases such as Dr. Windsor's of Mr. B. (p. 157), it obeyed plainly enough the periods of rheumatic aortitis. Stengel indeed speaks of febrile periods as well known in aortitis.[2] Popoff [3] also says, "Contrary

[1] Gilbert and Descamps, *Paris méd.*, 1912, vol. xvii. p. 044.
[2] Stengel, *Brit. Med. Journ.*, Oct. 20, 1906, p. 1011.
[3] Popoff of Moscow, *Zeitschr. f. klin. Med.* Bd. lxxv. He. 5 und 6, 1912.

to the usual opinion, aortitis is often attended with fever, though it may be too slight to attract attention if not watched for."

Gibson included as "by no means rare concomitants of anginous seizures," *vertigo, visual and auditory hallucinations*, and even *delusions*. Such symptoms we may observe not infrequently in aortic insufficiency, or arteriosclerosis, with or without angina; but not, I think, or not characteristically, in angina sole. However by many authors vertigo is mentioned as an incident of angina. I would add, as occasional prodroma, yawning and a vague sense of unrest.

It is for reasons such as these that I have placed *the vasomotor symptoms* in the secondary list (pp. 226 and 280), as incidental; in radical divergence from Nothnagel who, in an article written, it is true, forty years ago,[1] placed them in the first rank (*vide* pp. 224 and 236); whereby in my opinion he thickened the fog through which we have had "on each quarter to descrie the distant foe, Where lodged." Every fox-hunter knows how a flock of sheep may make his sport or mar it. By their movements a quarter of a mile off he may learn that the fox is passing their way; now the vasomotor nerves are these sheep. In some cases the stealthiest approach of angina pectoris may ruffle the watchers, and may startle them into an agitation under which the enemy may slip away unnoticed. But in angina this flutter, if it be caused by the enemy, is not the enemy. I have argued that under the irritation of any sensory nerve-trunk such vasomotor reactions readily appear, and the more as the centre becomes unstable; not only so, but in angina pectoris an emotion of them, like other, even subjective, causes, may by vasoconstriction determine the moment of a paroxysm. Still, this sympathetic disturbance is not of the essence of the malady, nor, as we have seen in discussing the pulse, is it a constant. In particular cases, or attacks, it may be difficult to disentangle a chance vasomotor commotion as a determinant of a seizure from one ensuing upon the distress of it; but the distinction is a real one, and in most cases is to be made out by the history of the seizure itself: was it instantly preceded by an annoyance, or a chill; or was it rather provoked by some muscular effort?

[1] Nothnagel, *Deutsche Arch. f. klin. Med.*, 1867.

The *complexion of the sufferer* does not tell us all that might have been expected. Many anginous patients are of turgid or weathered complexion, as some are spare and sallow; and the testimony is conflicting. Here again nature has no rule. Heberden speaks of pallor; Fothergill, a close observer of large experience, says distinctly that the face at first reddens, then in an instant turns very pale. Broadbent writes: "The face may be flushed or pale, or may keep its natural colour." John Hunter's face in his two attacks became pale and pinched. Of one of his own cases Sansom (*loc. cit.*) noted that in the attacks the radial artery contracted, and the face and mucous membranes turned pallid; the vessels then dilated, and the face and lips flushed. Sir James Goodhart,[1] in narrating a series of attacks, says, "His face flushed decidedly in the paroxysms." I saw more than once a subject of true and typical angina, who assured me, and her daughter was not less confident, that with the onset of a seizure her face flushed deeply, and the arteries of her neck began to throb; it was that as the attack passed away she turned pale. Nitrites increased the face flushing and made her altogether worse, as follows:

Mrs. W., æt. 60; angina pectoris; frequent attacks twelve months, especially after meals or on the least exertion. Previous health very good. With the onset (says her grown-up daughter) "her face always flushes, to turn pale when the seizure is over." When she feels the flushing coming on she is aware also of a throbbing in the notch above the sternum and in the neck, and the arm turns numb, without much pain in it; but the pain at the sternum is such that "if it didn't stop, her heart must cease to beat." Was it because of this vascular relaxation that, though the case was one of high pressure, nitrites made her feel worse and increased the face flush. The pressure, when she was as tranquil as one could expect, was about 200. There was a soft systolic murmur at the base. Perhaps our doses of nitrite were insufficient; some other areas of the periphery, *e.g.* the splanchnic, may still have been constricted.

Sir William Osler writes: "A sudden pallor of the face may be the first indication, and as a rule vasoconstrictor influences prevail in the severe paroxysms." In another patient, whose attack took place in his consulting-room, he reports: "The attack lasted a minute and a half; the face, normal at first as to

[1] Goodhart, Sir J., *Clin. Journ.*, April 4, 1894.

colour, then flushed deeply." But he adds, "Though not absent in the organic form of the disease, these vaso-constrictor disturbances are often more pronounced in the hysterical angina." In another paragraph he says, "The face may be death-like, ashen, or suffused or deeply congested even from the outset." In private notes of a certain case, sent to me by Drs. Mackenzie and Price, the complexion during the attacks turned "grey." Of another case Mackenzie [1] writes that "at the onset the face turned pale, and the radial artery became constricted and small" (see previous paragraphs). This case, like John Hunter's, of syphilitic aortitis, was made very interesting by the close observation of its clinical features. As the attacks came on, and they were almost incessant, the systolic pressures rose from a common level of about 160 to much greater heights—to 240, and, on one occasion at least, to 300. Among other interesting comments, Mackenzie suggests from his reading and experience that such fluctuations are especially frequent in aortic regurgitation, a malady in which a very powerful heart and an abnormal vascular adaptation are well-marked coefficients He thinks that the abnormal attitude of the vasomotor system makes it peculiarly liable to these extreme oscillations. My own recollection of such cases is in accordance with this suggestion. The author observes, in accordance with what I have said, that in some other cases of angina, in which he had made the same pressure observations, these rises of pressure with the pains did not occur. Gibson supported the general opinion that a deadly pallor is a frequent initial feature; but added that in his experience it had been "quite common to find the surface suffused by a bright flush."

Cyanosis is sometimes mentioned, but often no doubt in cases with dyspnea, and complicated with heart or lung disease. Profuse voidance of urine occurs in some cases after an attack, and suggests large areas of vasoconstriction other than renal. But, having already discussed these vasomotor phenomena under the head of mock angina, here I do but touch on them as subordinate symptoms of the true.

Dilatation of the stomach, by some process of sympathy or exhaustion, may be induced by angina pectoris; it is frequently

[1] Mackenzie, Jas., *Heart,* vol. ii. No. 3, 1911.

associated with it. Broadbent[1] and myself made this observation; and so before us did Elsner explicitly, but rather in respect of treatment—under which head I shall return to the subject—than as a point of pathogeny. It may be a consequence of vagus irritation, and we have noted that eructation often signifies relief. If then gastric ectasis is not to be reckoned as a correlated symptom of angina, the coexistence is by no means to be overlooked, as the mere oppression of it may provoke the recurrence of seizures, or even determine the moment of death. Dr. Verdon, in placing the seat of angina pectoris in the stomach —a hypothesis to be discussed presently (p. 344)—seems to me to exaggerate an important, but a secondary, element of this disease. One case, cited by Broadbent, of a distended stomach with a suspicion of angina, was a sequel of influenza; in this infection genuine angina is apt to ensue, but the interpretation is open to question. In one of Sir William Osler's cases—of a patient who died in status anginosus of fourteen days' duration—the author noted great gastric distension. The imminence of angina during digestion, whatever the explanation, is well known. As regards arterial pressures, these during digestion fluctuate a good deal. Usually there is an initial rise for a few minutes, then a fall on the whole for about an hour; then a rise of some duration. This excitement of angina on a full stomach can scarcely then be due to blood pressure. *Flatulency* has been regarded by all writers on angina pectoris as a general, a pressing, and perhaps a cardinal factor; but its significance is not easy to interpret. Dr. James Mackenzie has pointed out, with truth no doubt, that patients, attributing the distress to flatulence, belch and gulp in such a manner as to swallow air, and thus to produce or increase the very distress they apprehend. I have noted this point more carefully since, and think this explanation inconclusive. For instance, I noted this point in an elderly man whose first attack was severe and sudden. Setting aside some general arteriosclerosis, of which he had known nothing, he was well; nor was he beset by any dyspeptic symptoms. But, as this attack passed off, he said spontaneously that he became aware for the first time of flatulence, which rumbled up in "enormous quantities." Before

[1] Broadbent, W. H., *Lancet*, May 27, 1905.

it he had not belched or gulped, nor during it. His subsequent attacks were marked by the same sequence, although on my warning he took especial care to avoid every chance of wind-sucking. My own observations and those of others prove again and again that attacks of angina—of angina major or minor —often appear without any eructatory efforts, yet the wind and the pain may be discharged together. In some cases of angina pectoris swallowing is in abeyance. Now the suspicion of wind as the enemy is true not of angina only; it is one common to sufferers from almost all cardiac disorders, who suspect some correlation or bond between the wind and the heart's behaviour. Whatever the nexus, whether a vagus reflex from heart to stomach, or a mechanical butting at the heart itself, we are not yet in a position to say, everyday experience tells us that cardio-aortic maladies are mitigated by rules of diet which guard against generation of wind in the stomach, and are relieved by cordials which expel it when there. Dr. Verdon was a bold man when he passed an œsophageal tube during an attack of angina, an operation which might stimulate the lethal activity of the vagus; but the results in the two cases in which he tried it—one of angina pectoris, the other one of grave cardiac asystole—were no doubt strikingly successful.

Dr. Verdon's opinion, which in several well-known papers he has acutely advocated, is that angina has its seat in the stomach, and consists in an unruly gastric function which he defines at one time as "intra-gastric pressure" (distension ?), at another as "gastrismus" or "spasmodic constriction" of the viscus or its orifice. The author has examined the tender areas of skin and muscle, studied the intricate nervous connections of the stomach, and formed the opinion that referred pain in the eighth and ninth intercostal area signifies "hypertonus of the gastric muscle" (not "intragastric" pressure or distension ?). He reminds us of the several component strands of the vagus, of its distribution to the gastric muscle, of its radical association with the medullary portions of the spinal accessory, of the spinal connections on the cervical chain, of the association with the deep root of the musculospiral, and—at the seventh segment—with the ulnar deep root. Also he makes an interesting note on the salivation of some cases, which he traces through the chorda

tympani to the vagus. He illustrates his theme by the well-known complaints in angina of flatulence, loaded stomach, and so forth; associated gastric phenomena which, we shall agree, are found, not in angina only, but in almost all diseases and disorders of the heart? The close sympathy between these two viscera—partly nervous partly mechanical—and the great importance in therapeutics of realising these associations, Dr. Verdon has ably vindicated; but it is another and as yet an unverified postulate to assume that angina is not a cardio-vascular lesion aggravated by gastric reciprocity, but itself an outcome of no more than storms in the cup of the stomach. "Cardiospasm" is an old story; one of my earliest consultations was in a case of this kind, one which Mr. P. Teale helped to relieve by bougie. The attacks were relieved, but we did not put the malady to flight. Yet of all the many such cases of cardiospasm in my recollection not one suffered from angina pectoris. However I am glad to welcome Dr. Verdon as at any rate a disciple of my hypothesis of death in angina by vagus inhibition.

On the *physical signs* of the conditions congruous with angina pectoris I have little to say which is not common knowledge. Angina is prevalent among elderly and degenerated persons, and is apt therefore to be associated with signs of decadence; but the current opinion that the disease is always, and by its own nature, associated with widespread senile cardioarterial degeneration, is far from correct. The disease, as for instance we have seen in infectious aortitis such as the syphilitic or influenzal, is frequent in patients wholly free from either heart disease or arteriosclerosis. We have seen that the late Dr. Sansom, who for some years had shared the current prepossessions on this point, found himself obliged to revise his opinions; he frankly admitted to me that many cases had come before him in which neither hypertrophy of the heart nor thickening of the accessible vessels was to be detected. Sir James Goodhart (*loc. cit.*), after watching a series of paroxysms in a certain case, reports: "The heart was to all means of examination perfectly healthy, and upon a second examination I made this note: 'I am perfectly certain that if he went to an insurance office and said nothing, he would be passed as perfectly sound.'"

Disorder of rhythm, if haply concurrent with angina, is no character of it; in an anginous patient "gallop rhythm" may of course be casually present; but it is said that this character is never heard during an attack, that then it always ceases.

In the high-pressure cases the heart may be enlarged without other signs of disease. In uncomplicated angina, neither during the attacks nor between them is there any rule of dilatation or other static cardiac disorder. Is not the throbbing about the arch of the aorta, up into the throat, felt by many persons under excitement, as for example before rising to speak in public, sometimes enough to suggest not only a transient aortic dilatation, but even the sort of discomfort we are wont to associate with slight degrees of "stenocardia?" It seems certain that the arch may undergo considerable changes of diameter from vasomotor causes, as may the abdominal portion; and that these may be manifest as physical signs. Chafing or a cold sponge might reduce such an area of dulness. A very important physical sign, pointed out first by Péter, and often to be noted in angina, is the area of the aortic dulness already mentioned in the Section on Aortitis (see p. 198). We have seen that this area extends to the right of the manubrium, on the second space and third cartilage, forming more or less of the outline compared by Potain to the peak of a fireman's helmet. In normal persons submanubrial dulness is rare. And such dilatations may now be recorded by radiography. In two cases of my own we had this verification. The vessel may also be palpable in the episternal notch, and tender to pressure (see Mrs. X., p. 427); and the right subclavian may be elevated. Yet how punctilious we must be in taking comparisons at many points is illustrated once more in an important paper by Wenkebach,[1] where he argues that signs of dilated aorta may be simulated by a forcing up of the thoracic viscera by a high diaphragm. In such cases the heart may be bothered; but this bother is not angina pectoris. The dilatation of the aorta is most frequent and largest in certain syphilitic cases; in some of them so wide and the percussion note so flat as to suggest aneurysm or growth; but in some acute infections in young persons, as in rheumatic

[1] Wenkebach, *Nzderl. Tijd v. Gen.*, 1907 (translated).

aortitis, angina or no angina (p. 150), it is plain enough, *if sought for*. In some cases the aortic distension may be due, in part at any rate, to a paresis of the vessel, analogous to the paretic distension of an inflamed bowel; an interpretation which on recovery is often justified by the restoration of the aorta to its normal dimensions. Moreover during the same illness, as may be seen by radiograms, it may vary from one period to another. We have seen that aortitis is more frequent in acute rheumatism than is supposed, and in most cases is to be detected by physical signs only. Although not of the "muscular type," the aorta contains much muscle, and probably in health enlarges and contracts a good deal. Of Abram's reflex (*vide* p. 198) I have no practical skill, but I presume this method might assist us to distinguish static from dynamic expansions of the vessel.

When, as in chronic cases, the condition of the patient permits, before pronouncing that no signs of disease are present, the patient should be directed to perform some simple arm movements, as in dumb-bell fashion when, with an atheromatous aorta or valve, a systolic murmur, if not already present, may be elicited. On reference to my own cases I find that in the majority of them a systolic murmur was audible at the base. *In syphilitic cases even a slight alteration of the first sound at the base* must be regarded with grave suspicion, for a little muffling of this sound *often*—perhaps always—*precedes a murmur*. The suprasigmoid disease is then creeping upon the valve (p. 175), and the next event may be a murmur of regurgitation, when the case, if curable as angina, may as heart disease have become incurable. In such circumstances no vigilance can be excessive; no treatment too prompt. Dr. F. Jackson [1] discusses cases of his own in which disease just above the aortic valve, without perhaps invading it or the orifice, is marked by absent or very feeble sounds, even although the left ventricle be somewhat hypertrophied. "Such persons," he adds, "are liable to angina pectoris." In practice we are all familiar with the weakening of the first sound, and of the impulse (emphysema and fat chest wall excluded), which may signify a loss of vigour of the left ventricle, as in degeneration of the myocardium, or in anæmic or

[1] Jackson, F., *New York Med. Journ.*, April 28, 1894.

convalescent persons, or, as Bäumler says, in nodular fibrosis of the myocardium [1] (see p. 20). But so far from these signs being characteristic of angina pectoris, they are frequently absent.

Dr. James Mackenzie cites a case of angina (in a man æt. 42) in which signs of dilatation of the arch thus advanced to slight incompetence of the valve, and slight hypertrophy of the heart. The systolic blood pressures stood about 150, until, as his state grew worse and his sufferings more continuous, the pressure fell to 95. He died in a run of attacks sinking into heart failure. I have seen only too many such cases, especially in the syphilitic; but, with Mr. Balding of Royston, I saw two or three times a London merchant, aged 59, who six months before had begun to feel on exerting himself a tightness under the manubrium sterni; this grew worse, and a pain began to radiate into both shoulders, then down to both elbows, then to both wrists. If anything, the right side was the worse. Such was the dread of the attacks that he would take cab or bus to go but fifty yards from his office. The systolic blood pressure, under the ordinary circumstances of consultation, was not more than 140; and the accessible arteries were straight and soft. In this case syphilis was improbable, and on careful scrutiny no specific stigma was discovered. There were no symptoms of renal disease. Now his heart, as regards apparent size, position, and sounds, was normal, with one exception, which was this; there was a very distinct, almost musical, systolic aortic murmur; but the aortic second sound was not abnormal in quality. Our diagnosis was angina pectoris with subacute atheroma. As regards diastole, we did not forget how elusive an aortic regurgitant murmur may be; exceedingly soft and distant, especially so perhaps when first established, and then audible, or distinct, only perhaps in a small area to the left of the sternum. It is unnecessary then any longer to insist on close appreciations—of the quality of both first and second—of any undue stress, or alteration of it, whether of muffling, of reinforcement, or in what acousticians call "clang." The peculiar quality of the second aortic sound often present in these cases, and first observed by Gueneau de Mussy, I have described on another page (200).

[1] Bäumler, *Deutsche Arch. f. klin. Med.*, 1911.

The right subclavian may be palpable, as demonstrated first by Faure [1]; the radial pulses may be unequal.

I have said that in young persons an aorta dilated under acute inflammation, so as to yield obvious physical signs, may nevertheless pull itself together and recover, even *ad integrum*, or practically so; that in them the physical signs may diminish and even disappear; but in later life to the coats of a vessel which, as Professor MacWilliam, Herringham and Wills, and others have demonstrated, had been losing with time and wear its vitality and capacity of growth, such repair and restoration are less possible. On this point the reader may be referred to certain reflections of Jürgensen, in Nothnagel's *System*, on aortic insufficiency. In children however elasticity is not the factor chiefly concerned, for in them the arch of the aorta is relatively less rich in elastic fibres. Indeed there is some evidence that on the hither side of middle life the aorta may in case of need develop no little compensatory hypertrophy of a genuine kind.

I have one case on my notes in which the pupils were said to contract during the attacks. Permanently contracted pupils in angina, or the light stop, might, of course, suggest other terms of a syphilitic series, such as aneurysm, tabes, etc.

[1] Faure, *Arch. gén. de méd.*, 1874.

CHAPTER VI

HYPOTHESES OF ANGINA

After this summary consideration of the symptoms and signs, somewhat brief but sufficient perhaps for present purposes, we may proceed to discuss the interpretation of angina pectoris. This argument, which in some degree must take the form of controversy, falls into two parts; the criticism of those hypotheses which, whether for their own value or for the eminence of their advocates, still occupy the field, and an attempt on my own responsibility to substitute for them another hypothesis which, if it is to command attention, must explain more of the facts. Concerning the current conjectures, "theories" as some sanguine advocates call them, perhaps each disputant would be ready to admit that, his own excepted, they make an incoherent and motley collection. If, not united against a common enemy, they fell out among themselves, they would end in a mutual destruction—"Ceci tuerait cela." The heart cramp and the heart dilatation champions would come to a stalemate; and then the neurotic, the vasomotor, and the "pseudo" people, entering in and dissolving away the disease altogether, would make peace, on paper.

This incoherence was strikingly evident at a discussion on angina pectoris at a meeting of the Harveian Society in 1909, when once more I offered my own hypothesis.[1] Some speakers were clinging still to the orthodox view of coronary atheroma with thrombosis; others descried a ventricular distension, others a reluctance after the manner of an obstructive colic, others again a cramp of the heart; some invoked a spasm of the coronary arteries, or of the diaphragm, or of peripheral vascular areas; some, like Merklen, played two cards at

[1] Vide *Brit. Med. Journ.*, Dec. 11, 1909.

once, thus—*angina* is awakened "soit par la distension; soit par l'ischémie des éléments musculaires et des fibres nerveuses contenues dans leurs mailles"; others were vague, or wisely reticent. By a sagacious and discreet speaker the conclusion was fittingly summed up in these words: "Angina pectoris is a painful affection of the heart as a whole, depending upon disorganisation of some anatomical character, or a disorder of physiological function of one or more factors in cardiac action; and associated in some cases with a fear of impending death." That is to say, angina pectoris is anything you please —a very popular sentiment. But really, as Montaigne remarked on another matter, is there not more ado in interpreting the interpretations than in interpreting the things themselves? The late Professor Huchard is said to have collected and arranged no less than eighty hypotheses or, may we say, guesses, on this subject; I shall not follow his example. I must content myself with a smaller selection of the more prevalent conjectures, but these I will try to submit to a closer appreciation. I will consider the following opinions:

(i.) That angina pectoris consists in distension of an enfeebled ventricle (Parry, Stokes, Traube);

(ii.) That it consists in a cramp, or spasm, of the heart, analogous to cramp of a voluntary muscle (MacBride, Baumès, Latham);

(iii.) That it is an "intermittent claudication";

(iv.) That it consists in a myocardial ischæmia, generally due to an affection of the coronary arteries (atheroma, functional constriction, thrombosis, etc.), this *ischæmia* being the direct cause of the pain;

(v.) That it is "neural" in origin; a neuritis, a neurosis, a neuralgia, a vasomotor storm, local or general;

(vi.) That it is not itself at all, but a poor relation; something else of the same name flitting wistfully from one "entity" to another.

It is not, I trust, disrespectful to animadvert upon such a display of incongruous opinions as this; to reconcile such contradictions would demand a more than Hegelian insight. If the curability of a disease be inversely as the multitude of its remedies, so may our understanding of it be inversely as the

number of opinions about its nature. It will be pleaded, of course, that no one holds two of the incompatibles at once. Well, perhaps not in the same breath; but the hard-pressed disputant flies for refuge now to one, now to another; yet to bolster up one hypothesis by another does not prove either, still less both. To quote examples would be invidious, and perhaps not very useful. Can we indeed avoid an apprehension that the vogue of such a competition of promiscuous guesses betokens not a defective knowledge only, but also on this subject an undisciplined state of medical ideas? That so heterogeneous a mob of conjectures should engage our attention, that one of the ablest physicians and teachers of the day should in a standard treatise vacillate between such vague speculations as partial spasms of the myocardium; a cramp of the heart comparable with renal, hepatic, or intestinal colic; a stretching of the ventricle, and "claudication," seems to argue a defective sense of proportion, of comparative values. And yet, notwithstanding, I am about to press into the ragged regiment a recruit of my own.

The older writers, Bonnet (*Sepulchretum*), Morgagni, Heberden, discovered in their necropsies on angina morbid states so many and so various that naturally they were perplexed. For any or all of these states were more frequent without angina than with it. But from Jenner's time attention was riveted on the coronary arteries. It is true, no doubt, that an assumption of the invariable association of coronary disease with angina, as first suggested by Jenner and Parry, is compatible with the attribution of the several attacks to some intermediate factor, such as a ventricular stress, or a cramp. For this reason I will begin my scrutiny of the pathology of the disease by examining the implication of these arteries in the process.

In the later eighteenth and early nineteenth century there was much scepticism concerning the dependence of angina upon coronary disease, but of late years the coronary hypothesis has been urged with an almost blind advocacy. Our fathers took note both of coronary disease without angina, and of angina with coronaries intact.

Coronary Disease. — It is freely admitted of course that

coronary disease may, and indeed for the most part does, pursue its silent way without anginous or other storms; and that, while angina is most frequent in later middle life, coronary disease extends, as years extend, into decrepitude. It is to be regretted therefore that modern authors should rehearse the really tiresome burden of "coronary angina," "angine coronarienne," "die der richtigen Koronärsklerose entsprechende Angina," and so forth; as if the two conceptions, that of coronary disease and of angina, were indissoluble, or at any rate twin; or as if on coronary disease depended the mutation, the functional swerve, the "unhallowed string which vilely jars." "True angina," says one of the more eminent, "always presents lesion of the coronary arteries; this is its anatomical substratum." Neuburger [1] thinks he clinches the argument by the necropsies of thirty-eight cases of mortal angina in every one of which he found some degree of disease in the coronary arteries; it appears that all these cases were in old people, yet of the ordinary incidence of coronary disease in elderly people he offers no estimate. Von Romberg, even in his last edition (p. 43), still confidently assumes "the origin ('Entstehung') of angina to lie in coronary sclerosis"; and conversely that this pathological change is the cause of angina. So Krause likewise, in his new *Lehrbuch*, attributes "angina vera" to disease of the coronary arteries mainly, if occasionally to myocardial degeneration not so caused. Schmoll,[2] with an assurance no less ready, formulates the maxim that angina pectoris is in proportion to the cardiac disease due to coronary atheroma, and bids us rest our prognosis upon the degree of this lesion. Balfour, on the contrary, pondered over the strange inconsistency that such conditions as these are usually unattended with angina. Unfortunately in cases of angina necropsies are difficult to obtain, at any rate in private practice; the death is usually sudden, the occasion fleeting, the household distracted, and the diagnosis sure enough to warrant a certificate.

Now against the assumption that angina depends upon coronary disease a cautious writer would recognise no little

[1] Neuburger, *Deutsche med. Wochenschr.*, Juni 13, 1901.

[2] Schmoll, "Über mot.-sensor. u. vasomot. Symptomien in Koronärsklerose," *Münch. med. Wochenschr.*, 1907, No. 41.

evidence, direct or indirect; not only from the older authors, such as Desportes,[1] Jurine, and others, whose pathological equipment might be regarded as rough, but also from modern pathologists. Fothergill[2] noted disease of the coronary arteries in angina—they were diseased in the typical case of a man, aged 65, whose body John Hunter dissected for him—but Fothergill does not seem to have attached any special significance to this coincidence; he suspected rather that the cause was obesity about and within the thorax. Gibson says indeed that, although Jenner was the first to correlate coronary disease with angina, Fothergill was the first to publish the coincidence.[3] Then the ossified coronaries were supposed to offer a mechanical hindrance to the heart's motion; but soon, by Parry and others, it was seen that in this case the nutrition of the myocardium would suffer, and blood gather in the cavity. Parry and Matthew Baillie took the coronary hint from Jenner; and Peter Joseph Frank[4] half agreed with them; but they noted exceptions to the rule, and thought the mechanism of the pain to be not, as Heberden supposed, a spasm of the heart's muscle, but an oppression of it by this congestion of blood in the chamber. Desportes shrewdly demurred that, having examined many bodies of persons over 50 years of age, he found coronary atheroma "at least as common" in the women as in the men, yet that in women angina was rare. How far coronary atheroma may be the cause of angina or not is an open question; but when it is asserted that Jenner demonstrated, or declared, that angina depended upon their decay we exceed historical testimony. In his Letter, it will be found that Jenner, with true scientific caution, did not attribute the angina to the coronary changes; he stated simply that after death in angina disease of these vessels would be found; another and a different proposition. To point out the significance of coronary disease in angina was true service, but for his successors to force his meaning, and to declare coronary atheroma to be the immediate cause of the attacks, is to pass, where he did not, *ex errore per veritatem in errorem.* Jenner was speaking of course from the

[1] Desportes, *Traité de l'angine de poitrine*, Paris, 1811, an excellent book.
[2] *Loc. cit.* p. 72.
[3] In the *Med. Obs. Soc. Phys.*, London, 1776, vols. ccxxxiii. and cclii.
[4] Frank, Peter J., *Praxeos med. praecepta*, Lips., 1824.

evidence of mortal cases only; if angina kills people, then coronary atheroma will be found, a true point, to be taken presently. Everard Home reinforced Jenner's emphasis upon the coronary disease noted by Heberden and others in cases of (mortal) angina, by attributing the pain to the pressure of vessels thus hardened upon the ramifications of the cardiac nerves, a possibility hinted at again by Dr. Morison in discussing a section (p. 404) taken by him from the heart substance in a certain case of angina, in which a small intramyocardial aneurysm was seated pressingly near a nervous ganglion and fibres.[1] But, as we shall see later, these myocardial ganglia are pretty surely motor, not sensory structures.

Now Cohnheim says: "Often no trace of disease is to be found in the coronary arteries of these subjects of angina." Indeed in one of Heberden's cases the coronary arteries were unaffected, the only lesion discovered being atheromatous patches in the aorta. In John Hunter's heart the coronary arteries, though "ossified"—"they cut with difficulty, and gaped open when severed" (Home)—seem to have been pervious; and Jores has pointed out that ordinarily in sclerosis of the trunks of these vessels the intramyocardial twigs are unaffected. Dr. Morison, when publishing[2] a case of angina in which atheromatous changes in the coronary arteries were in a comparatively early stage, and the orifices normally patent, ventures so far as to hint at some dissatisfaction with "this ready coronary explanation." Before turning to my own evidence, which may be biassed, let me cite further that of others. Fränkel says (in the fourth edition of Eulenburg): "Angina arises also in other heart diseases; for instance in certain valvular affections, and under conditions where the coronary arteries are intact throughout ('sich als durchaus intakt erwiesen')." This is the testimony of an opponent; for, in the dilemma, this scrupulous observer is driven to assume that cramp of these vessels which we shall see to be a mere guess, and a guess contrary or, at any rate, alien

[1] Morison, A., *Cardiac Pain*, 1902.

[2] Morison, *loc. cit.* (*Cardiac Pain*). This careful writer adds that there was nothing in the aorta to account for the angina; but he did not bring the investigation he gives to other structures to bear upon this one. Minute sections were made of the myocardium, but no sections of the aorta; here a glance of the naked eye, and probably at the inside only, is too often sufficient.

to what we know of their nervous endowments. Hirsch [1] freely admits that most cases of coronary sclerosis take the course of chronic heart insufficiency without any symptoms of angina, and in these cases he finds amyl nitrite to do more harm than good. Thorel, in his review of "Angina Pectoris" in *Lubarsch* for 1907, says the interpretation of the disease as a myocardial ischæmia is far from proved; for life is often prolonged with both of these vessels closed, yet without angina. There is, he adds, some factor of this disease as yet unperceived ("ein unerklärliches Etwas"). As to the fibroid nodules resulting from coronary disease, we may find, he says, even with these masses in abundance, that conductivity is quite unimpaired. Dr. Samuel West [2] reports a case in which one coronary was completely obliterated, and the orifice of the other so minute that no blood could have traversed them to the heart, yet without angina. Conversely, in many cases of angina pectoris the myocardium has proved to be perfectly normal, even without interstitial fibrosis, as in cases examined by Gennari.[3] Balfour, as I have said, frequently returns to these difficulties: for instance he writes: [4] "Coronary sclerosis is too often present when there has never been any angina to permit the occurrence being looked upon as anything more than accidental. . . . These arteries, being unsupported, are very liable to stretch and twist." Again he reflects: "Like arteriosclerosis, fatty degeneration of the myocardium is too often found when there has been no antecedent angina . . . to allow us to regard the association as more than a concomitant." He then drifts into speculations on imperfect metabolism, cardiac reserves and so forth.

To cover the many exceptions, and to get over these difficulties, a *spasm of the coronaries* has been postulated, an argumentative resource first supplied, I think, by the ingenuity of Neusser. Thus one influential author speaks to us of "spasm of the coronary arteries causing high vascular tension within the myocardium," a process I have tried to picture to my mind in vain. He is more intelligible when he adds that the temporary ischæmia thus

[1] In Penzoldt and Sterzing, new ed., vol. iii. p. 372.
[2] West, S., *Lancet*, Oct. 16, 1909.
[3] Gennari, *Arch. pour les sci. méd.*, déc. 1905, and p. 17 of this volume.
[4] *E.g.* Balfour, *Senile Heart*, p. 127.

produced provokes a spasm of the heart muscle. Yet, on the other hand, Wooldridge and others have shown that a transient myocardial anæmia is no very grave matter. In a recent authoritative work it is said positively that "nitroglycerine is used to dilate the coronary vessels in angina pectoris." Sheer guess work! Ortner talks, as if they lay visible before his eyes, of forcing blood into the constricted coronaries by means of digitalis or theobromine; and of combinations of vasodilator drugs with digitalis in order so to open out these vessels that digitalis may pump blood in. It takes some presence of mind to realise that these convenient "*facts*," stated so calmly and so confidently, are at best opinions, and hitherto sheer fancy! How can a spring up three stairs, an effort at stool, or to walk against the wind, constrict the coronary arteries? It should dilate them; pretty surely it does. And more plodding observers might suppose that myocardial anæmia or "ischæmia" would betray itself by a fall in blood-pressure, a severe fall if produced by a prolonged or extensive coronary closure. Now although, as many authors agree [1] (*vide* p. 335), the frequency of high arterial pressures in angina, recorded by Brunton and later by Pal, has been exaggerated, yet it is also agreed that persistently high pressures are consistent with the disease; and sometimes run very high. Indeed, the opinion prevails, however inconsistently, that the relief by nitrites, and empirically this relief is obvious, depends upon an abatement of these arterial pressures. In cases of broken compensation, said Broadbent, perhaps a little too positively, angina pectoris is unknown. Again, how is it, if the coronary arteries are endowed with these vermicular activities, that they do not more often put them in practice? Why, if this be their way, are not casual whiffs of angina a common experience? Vasoconstrictions, extensive and partial, are otherwise common enough; but if the coronaries were prone to participate in them the heart would be at a double disadvantage; with rising peripheral resistance its reserve might be cut off: fortunately, as we know, peripheral vaso-constriction ordinarily means coronary dilatation. Yet even von Basch surmised that angina pectoris may be caused by cramp of the coronary arteries; Huchard perpetually

[1] Mackenzie. Jas., *Heart*, vol. ii., 1911.

invoked it and its alleged consequence of "increased intracardiac pressure" (!); and no less a man than Balfour dallied with the notion. The explanation is so handy it cannot but be true! We are now assured, by Ortner for instance (*loc. cit.*), that in the trembling balance of life, spasm may be as well or better postulated of diseased than of normal arteries, a contention which may have some grains of truth in it. He betakes himself to the cramp hypothesis because "we see not rarely very severe angina pectoris *with coronary changes minimal or absent, and their orifices clear*" (italic mine); and he finds no proportion between the degrees of angina and of coronary lesion. Yet coronary it has to be, somehow! That the life of the healthiest of us should be at the mercy of a squall in so narrow a belt of arterial irrigation, whether in the heart or in the bulb, is disquieting enough, but what is this to a hypothesis! The truth is, spasm of the coronaries is a nosologist's conceit to explain puzzles of his own making.

How far in sclerosed arteries the capacity for spasm may be presumed to survive, or indeed how far in disease it may be even in morbid excess, and, if so, how this may be mechanically explicable, I have discussed elsewhere (Arteriosclerosis, p. 162); suffice it, in respect of the coronaries, to say that in the first instance it must be proved that in any conditions these vessels are dominated by vasomotor governance.[1] Heymans and Demoor,[2] in their comprehensive research into the innervation of the myocardium, Professor Brodie and Miss Cullis,[3] and other observers have, it is true, detected both vaso-constrictor and vaso-dilator activities in these vessels; but Professor Schäfer, in the course of a large experience with adrenalin, concludes that they have very little contractile power; and, generally speaking, physiologists are of the opinion that although such agents as adrenalin affect their muscular coat directly, usually indeed to dilatation,[4] yet it is but slightly, and their tides are determined by the oxygen tension—and of course by the blood pressure. Meyer is of opinion [5] that although

[1] See Langley, *Journ. of Physiol.* vol. xxxii., 1905.

[2] Heymans and Demoor, *Arch. de biol.*, Gand and Leipzig, 1893–95, vol. xiii. p. 619 (25 plates, etc.).

[3] Brodie and Cullis, *Journ. of Physiol.* vol. xliii., 1911. [4] See Vol. I. p. 230.

[5] Meyer, F., *Berl. klin. Wochenschr.* vol. l., 1913, p. 920.

after injection of adrenalin the coronaries expand, under more heart work and higher pressure, yet a dilating effect due to the adrenalin is also apparent. It is not easy to see how Meyer can discern this difference, and he seems to me to neglect the increase of the nutritional and oxidising activity. Langendorff had proved already [1] that, when by adrenalin an extensive vasomotor constriction is set up, these vessels expand to support the heart in the increase of its work; their feeble innervation could not, and ought not, to withstand a systemic rise of pressures, or whence would come the heart's needed increase of energy? Mr. Barcroft has demonstrated that coronary flow, if modified at all, is not regulated by vasomotor governance; that their diameter follows the amount of CO_2 given out by the cardiac muscle, or the correlative metabolism. As he illustrates by carefully constructed charts, we see that if the heart be deprived of oxygen by fractions—*e.g.* O_2 being as 1·0, 0·6 . . . 0·2, 0·1 per unit time, the curve of coronary flow does not droop in parallel with the oxygen time, it is maintained a little longer; and then falls towards it by a more rapid gradient.[2] Dr. Cow,[3] employing Meyer's method, found, in accordance with other observers, that although adrenalin constricted the arteries of many areas, it did not constrict the intravisceral branches of the coronary, pulmonary, and cerebral arteries; indeed the coronary and cerebral were dilated rather. Moreover, pursuing the researches of Barcroft, and of Dixon, he found that CO_2, as correlative of cardiac metabolism, dilated the coronary (as also the carotid, gastric, etc.) arteries. The gas was bubbled through the Ringer's solution; as the gas was turned on and off the coronary arteries responded accordingly. The effect was direct on their vascular coat, not on nerve-endings. Dr. Argyll Campbell testifies to the same effect.[4] Dr. Laidlaw [5] says the influence of the heart-beat on the coronary flow is profound; but I should hardly say "profound," the pressure and the myocardial oxidation are dominant. An increased rate

[1] Langendorff, *Zentralbl. f. Physiol.*, 1907, No. 7.
[2] See also Morawitz u. Zahn, *Zentralbl. f. Physiol.* vol. xxvi.
[3] Cow, *Proc. Roy. Soc.*, Feb. 20, 1911; and *Journ. Physiol.* vol. xlii., 1911.
[4] Campbell, Argyll, *Quart. Journ. of Exp. Physiol.* vol. iv. Nos. 1 and 2. See also Laidlaw, *Journal of Physiology*, vol. xl., 1910, p. 487.
[5] Laidlaw, *Biochem. Journ.* vol. vi. No. 1.

of beat causes a quicker coronary outflow *without dilatation of the coronary vessels* (italics mine). As I have said, if the coronaries took part in extensive vaso-constriction the heart would not be left wholly free to liberate energy to meet the rise of arterial pressures. These arteries have developed superficially in order that they may be thus passive to the blood-flow; were this free play impeded by constrictions the coefficient—more heart work, more blood, would be inconstant. Aschoff [1] infers that because the coronaries are rich in muscle therefore they are the more subject to nervous influences; but this by no means necessarily follows: the strong muscular tone may as well be to protect these vessels against the dilating stresses of sudden increases of demand. The prevalence of atheroma in the coronary arteries suggests notwithstanding, that, under the frequent calls upon output, these vessels do suffer under lateral stresses, against which vigorous automatic tone tends to protect them. Huchard, in his airy way, said that the coronaries, being near the heart, are therefore under the highest pressures. Not so; although their mouths are bell-shaped, yet the angle of their bifurcation is sharp, their size is small for the work they do, and especially small for their intricate peripheral bed. The velocity is relatively high, but the friction is high also. Still, as Roy pointed out (*Phil. Tr.*, 1892), an increase of pressure in these vessels multiplies the cardiac energy enormously. Coronary supply has however little effect, if any, on the rate of the pulse. Let us hope then that our coronaries are incapable of such perilous cantrips, and postpone consideration of spasm of these vessels, whether in health or disease, whether in real angina or sham, until we have proof. Whatever the motor energy of these vessels, it is towards dilatation (sympathetic); for constriction (autonomic) it is very slight.

It is natural to assume that if the coronary arteries are diseased the myocardium must suffer. Complete obliteration of the coronary trunks during life certainly often happens, yet, strange as it may seem, if a very chronic process, as it usually is, the heart may in spite of it stick to work long enough. Sir William Osler has said:[2] "A man may get on very comfortably with . . . practi-

[1] Aschoff in a discussion reported in *Deutsche med. Wochenschr.*, Feb. 20, 1913. Morawitz u. Zahn in the same discussion laid emphasis chiefly on the blood pressures.

[2] Osler, Sir W., "Lumleian Lectures," *Lancet*, March 26, 1910.

cally a fourth of the whole (coronary) system" (if, that is, the reduction be very gradually established). Steven[1] also observed: "The amount of deprivation of nourishment which can be borne by the heart, if it be slowly induced, is wonderful." Dr. West (*Path. Trans.* 1884) has said the same thing; and W. H. Dickenson testified (also in *Path. Trans.*) that "closure of the coronary arteries does not prevent even considerable hypertrophy of the heart." I have discussed a part of this problem already in the chapter on Cardiosclerosis (p. 21), and pointed out that "a man may get on" with the whole system occluded, so far at any rate as the orifices and main trunks are concerned.

The effect of coronary disease upon the myocardium was discussed by Morgagni, in a well-known chapter of his great work, and by Cruveilhier.[2] Virchow and his school returned to the subject, and Huber[3] showed, as the French school has shown, that the resulting fibroid changes, unless periarteritic, were not inflammatory, but "dystrophic sclerosis" (p. 23). Cohnheim[4] said that callus and fibrosis (p. 21) ("sometimes a host of callous bodies scattered through both ventricles") do not betray themselves by any symptom whatever; and that the same was true of more diffused "myocardial indurations." It seems that, so long as there is no heart block, contractility is but little affected. Although, as I have said, decay of the myocardium is not a uniform nor a universal result of coronary obstruction, every physician must of course regard the state of these vessels as important in so far as the nutrition of the cardiac muscle may suffer in consequence. Yet Fränkel and many others admit, what Kanthack and I demonstrated, the frequent immunity of the cardiac muscle from degeneration, even in obliteration of the coronaries,[5] or at any rate of their orifices—as Fränkel says, the muscle is often normal ("intakt befunden")—and this is explained, probably with truth, by supposing some considerable tracts of these channels to remain, and to receive blood from other sources.

[1] Steven, L., *Lancet*, 1897.
[2] Cruveilhier, *Traité d'anat. pathologique.*
[3] Huber, *Virchow's Archiv*, Bd. lxxxix.
[4] Cohnheim, *Syd. Soc.*, ed. 1889.
[5] I learnt afterwards that we were not the first to arrive by this way at the same conclusion.

Kanthack and I concluded that, other things equal, the factor of safety is the rate of occlusion; if very slow an alternative nutrition may be established even when the orifices of the coronaries are so overgrown that they cannot be detected.[1] Dr. A. Morison[2] has also testified to this remarkable independence of the myocardium of the coronary main trunks or mouths; in a carefully observed case, in which the coronary arteries were almost completely blocked, he found the muscular tissue of the heart if in part fibroid yet "on the whole well preserved." He makes the general statement that a sound myocardium may concur with extreme coronary occlusion; and on the other hand that much degeneration of the cardiac muscle may go forward while the coronary arteries are open.

Cabot[3] rightly emphasises the importance of comparing clinical and pathological reports independently of each other; and in respect of angina and coronary disease he compared such reports independently. He concluded that in "patients with well-marked angina pectoris and clear evidence of a general arterio-sclerosis," in whom accordingly coronary obliteration or narrowing and consequent "fibrous myocarditis" were confidently assumed, he had often been convinced on the plain testimony of post-mortem examination that the pathological part of the diagnosis had been wrong. More refined methods of injection have proved that the coronaries are not "end-arteries"; their smaller branches anastomose freely everywhere in the structures to which they are distributed; not only so, but cross connections between arterial and venous branches have been demonstrated. Dr. West, Dr. Aldren Wright, Orth, and others (see Spalteholz, p. 23) verified Haller's[4] observation of the freedom of coronary anastomosis; and injections of red lead and gelatine made these communications plain in the X-ray pictures shown at the discussion at the Congress for Inner Medicine held at Wiesbaden in April 1907.[5] They were verified also by Janin and Merkel's beautiful stereoscopic skiagrams showing anastomoses between branches of no inconsiderable size. Some of these

[1] *Vide* chapter on Cardiosclerosis, p. 23 of this volume.
[2] Morison, A., "*Treatment*," Oct. 28, 1897.
[3] Cabot, *Massach. Gen. Hosp.*, Oct. 1911.
[4] Haller, *Elem. Phys. Corp. Hum.*, Lausanne, ed. 1757, p. 137.
[5] And again at the International Congress of 1913.

indeed, as Professor Spalteholz has shown to me, are visible to the naked eye. Moreover, Hirsch described some skilful experiments upon dogs and monkeys by which he proved that ligation of large coronary branches, *e.g.* the descending branch of the left coronary, at various levels, if they impaired, did not seem to abolish the functional capacity of the heart in these animals, experiments which seem to contradict clinical experience in man; but in man muscle and vessel, and the whole complex of function, may be in more delicate balance; and animals under experiment are not exciting themselves. I have quoted the recent observation of Jores that in many cases of atheroma of the coronary arteries, if not in all, their intramuscular twigs are not affected; a statement largely true of the vessels within some other viscera also, such as the liver.

Marchiafava [1] has divided coronary disease and its effects into five varieties, namely: (1) Simple sclerosis without grave alteration of the myocardium; (2) Sclerosis with acute or chronic lesions of the myocardium; (3) Stenosis of orifices, without vascular sclerosis or myocardial changes; (4) Old obliteration of one large coronary branch without myocardial disease; (5) Obliteration of one large coronary branch with infarct or myomalacia, and with or without rupture of the heart. He discusses the cardiac consequences which I have indicated summarily; my purpose at present is only to show that as coronary disease, even closure of both orifices, if initiated so slowly that alternative channels, or alternative sources for the old channels, can take up the supply, need not, and often does not, bring general myocardial degeneration in its train. Dr. Cowan,[2] in a summary of such cases, points out that if one coronary be blocked, the other may hypertrophy to supply its place; an observation, by the way, which affords additional evidence of their confluence. But the effects of block seem better marked in the left than in the right coronary. Pratt suggested that the heart was nourished by the veins of Thebesius, but they open on the right ventricle where the blood is blue. Redwitz, of the Pathological Institute of Munich,[3] likewise compared

[1] Marchiafava, *Riv. critica di clinica med.*, April 13, 1904.

[2] Cowan, John, *Trans. Glasgow Path. and Clin. Soc.*, 1902.

[3] Redwitz, von, "Erkr. d. Koronärarterien u. chron. Aortitis," *Virchow's Arch. f. path. Anat.*, 1909, Bd. cxcxvii.

cases in which the coronary arteries were obliterated with those in which they were permeable, or even normal, but blocked at their orifices by aortic disease; in this case they contained blood nevertheless, and the myocardium kept fairly good. He thinks the callous lumps are not direct consequences of coronary atheroma, but are a consequence with it of some common cause, as yet unrecognised (p. 21). Yet, with the looseness with which our clinical labels are distributed, the author proceeds to enumerate among other consequences of coronary disease, "cardiac asthma, or angina pectoris from pulmonary œdema"! It would be difficult to get more blunders into eight words. Broadbent[1] admitted that angina might occur before coronary disease had brought about any recognisable changes, before even microscopical blur of the muscular striæ. He persisted nevertheless, on slender grounds as it seems to me, that in angina "the condition of the heart walls must be the dominant factor," and elsewhere he speaks of it as an indispensable factor: now the dominant factor in the mortality of angina it is. Even Nothnagel admitted latterly that with the angina itself the myocardium has little to do. In all cases then, if used as facts for argument, the myocardial condition must be precisely defined.

The following cases, out of forty collected by Dr. Aldren Wright,[2] may serve to illustrate the comparative independence of coronary disease and apparent myocardial degeneration. Of twenty of these, 300 specimens were cut and stained.

I. Male, æt. 56. Angina pectoris. Left coronary closed; right artery pinhole. Cardiac muscle of good colour and firm. Micr. no fatty or other change.

II. Old man, died in attack of angina. Right coronary artery admits bristle; left coronary artery, "much narrowed." Micr. "Slight fatty degeneration," which "needed some search" to detect it.

III. Male, æt. 42. (Angina pectoris, probably syphilitic.) Died suddenly. Left coronary artery and right coronary artery barely admitted probe. "Myocardium quite healthy."

In three other of Dr. Wright's cases, in which with angina the coronary orifices were occluded, or nearly so, of the myocardium

[1] Broadbent, W. H., *Lancet*, May 27, 1905.

[2] Wright, Aldren, M.D. thesis (Cambridge), 1902. "Occlusion of the Coronary Arteries and its Effect upon the Heart."

"Hypertrophied and slightly fatty," "Slightly fatty," and "Appeared normal but for a little fibrosis here and there" were reported respectively. Of course in many of his instances of coronary occlusion the myocardium was found decayed, but in no correspondence with a history of anginous symptoms. In some of the more normal hearts he noted that the fine vascular network was particularly numerous and open. In the large majority of his cases of coronary disease Wright, like West,[1] found no history of angina.

I will add, by way of broadening my testimony, a few more cases by other observers.

IV. Notes of a case sent to me by the kindness of Dr. Bennion of St. Mary Cray. Male, æt. 43. Angina pectoris case entered during his time at St. Bartholomew's. At death, heart 13 oz.; left coronary artery "much narrowed"; right coronary artery, "a slit." Micr. "Cardiac muscle normal." "Aorta atheromatous and calcareous; large atheromatous ulcers in places." Dr. Bennion states: "The autopsy was careful, suspicious, and complete, yet the musculature of the heart was proved not to have suffered."

V. Male, æt. 45. Angina for three years, died in attack.[2] Heart large and heavy; left coronary artery closed by atheroma, vessel collapsed. Right coronary artery much narrowed at orifice but pervious, and otherwise retaining its full dimensions. Coats of both free from atheroma. The atheroma had encroached from disease of the root of the aorta, where it formed a soft cushion under the lining membrane. Myocardium "slightly fatty, but not more than most hearts are found to be."

VI. Male, æt. 42. Soft cushion-like atheroma at the root of the aorta, under the lining membrane, encroaching on the mouths of the coronary artery, so that neither would admit the probe without some pressure. Heart 13½ oz., colour and texture natural. Micr. "trifling diffuse fatty dotting, but not such as to indicate any morbid change."

VII. Practically the same report. Myocardium normal. Coronary arteries: right coronary closed; left narrowed to about size of probe. [In referring to these three records, I was much interested to see that the *authors pointed out that coronary disease was less characteristic of angina than atheroma of the beginning of the aorta,*

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[1] West, S., *Path. Trans.*, 1884.

[2] Cases V.-VII. are taken from W. H. Dickenson and Dudfield, *Path. Trans.* vol. xvii., 1865, "Angina with Coronary Disease but without Cardiac Degeneration."

and about the valve; and that they disagreed with the cardiac spasm hypothesis. Cases V. and VI. were probably syphilitic.—C. A.]

VIII. A like case (probably syphilitic) recorded by Balfour (*loc. cit.* p. 324).

IX. A like case, described by Bartholmès, in an Erlangen thesis of 1901,[1] as follows: A powerful man, æt. 35, ill six months. In heavy labour as an ironfounder. Moderate in alcohol and tobacco. "No syphilis." Angina pectoris of great severity, culminating in status anginosus and death. "Aortitis of ascending aorta." Coronary arteries extremely narrow throughout, so much so that in their uniform narrowness the author considered them to be unique. Of the cardiac muscle minute histological examination was made, with negative result. "All the fibres were normal"—to all tests—and interstitially the only intrusion of fat was in very slight degree about the apex.

X. Male, æt. 24, "died of angina pectoris with a practically healthy heart." A ring of thickening occupied the whole circumference of the aorta at and beyond the sinuses of Valsalva. The intima was swollen to twice or thrice its normal thickness, with a clear translucent deposit. (Obviously syphilitic.—C. A.) The openings of the coronary arteries in the midst of this area were so contracted that a probe could scarcely enter. *The cardiac muscle, however, was of a good colour and consistence and microscopically its structure was normal* (italics mine).

XI.-XII. One of Porter's cases.[2] Male, æt. 56. Angina Pectoris. Left coronary orifice occluded, right a pin-hole. Myocardium good colour and firm. No fatty or other degeneration.—Another case is cited of an old man who died in an attack. Right c.a. admitted a bristle; the left was narrow. Only on close search was a rather fatty state discerned. (See also Mackenzie's case, p. 434.)

Dr. Aldren Wright found that aortic disease had encroached upon the coronary orifices in 28 out of 40 cases.

Dr. Poynton[3] mentions a case of syphiloma of the heart, attended with nearly complete obstruction of the coronary arteries, in which the myocardium was quite normal. Dr. Mitchell Bruce, in a review of this aspect of the argument, says:[4] "My observations confirm Sir Clifford Allbutt's statement, that in syphilis of the aorta the heart below the aortic

[1] Extract in *Virch. Jahresb.*, 1902.
[2] Porter, *Amer. Journ. Exp. Med.*, 1898.
[3] Poynton, *British Medical Journal*, March 1900, p. 635.
[4] Mitchell Bruce, "Lumleian Lectures," *Lancet*, July 8, 1911.

valve, save for some endocardial opacities, is healthy." Dr. Morison says decisively that open coronary arteries occur with myocardial fibrosis, and closed coronary arteries occur without it. Fränkel has rather reluctantly testified to the same effect, and clings to a supervenient spasm of these vessels. But, as Neusser[1] perceived, are we to suppose that with increasing rigidity these vessels become more and more ready to contract? However, we must admit that the muscle, normal as in structure it may appear, must be more or less short of food, and so far embarrassed in function. The fair-seeming muscle, as in Dr. Anderson's elderly cat (p. 475), is doubtless impaired.

Suprasigmoid aortitis is so prone to involve the mouths of these arteries that crucial cases of angina, with coronary orifices intact, are the less easy to discover. However, we see there is much evidence that coronary stenosis is not necessarily followed by visible myocardial degeneration, and in alleging coronary obstruction as the cause of angina we must strictly enquire whether these orifices were absolutely blocked or narrowed only.[2] In many case reports of angina we observe that a partial coronary occlusion satisfies the reporter, and not unreasonably; but this admission cuts both ways. If through minuter orifices the myocardial nutrition can still under hydrostatic pressure be kept up, why the angina? Because the cardiac reserve is less? Then what of the multitude of cases of obvious myocardial degeneration without angina?

When witnesses so trustworthy admit that angina may occur before coronary disease has brought about any visible changes in the myocardium, when not even a microscopic confusion of the striæ can be detected, and yet in the same breath protest that "in angina the state of the heart walls must be the dominant factor," do not we overhear an echo of Vesalius's final submission to the dogma of desudation of the blood through the pores of the ventricular septum? And when, on the other hand, in the fourth edition of Eulenburg Fränkel concedes that in some cases of angina the coronary arteries are proved after death to have been sound—*durchaus intakt*—and yet clings to

[1] Neusser, E., *Symptomatologie u. Diagn.*, H. 2, 'Angina P.', Wien, 1904.

[2] Atheroma, as contrasted with occlusion by encroaching aortic disease, in the coronary arteries most frequently begins in the anterior vessel, about one inch from its orifice.

the coronary hypothesis, are we not listening again, not to the man who "treasures his exceptions," but to one whose judgment is subjugated to an orthodox dogma?

Let us now enquire if a block of one of the coronary arteries, or a rupture of one of them, with bleeding into the myocardium, causes pain. It is true that a sudden thrombosis is often attended by intense pain so like angina that we cannot refuse it the name. It is fierce, and in site substernal, though generally more continuous. To this event we shall return. But that mere reduction of the blood-supply does or can produce severe pain, a maxim attributed to Allan Burns,[1] and more or less favoured by Charcot[2] and by Potain,[3] seems to be taken for granted; though our common experience of coronary atheroma and of the various anæmias indicates much to the contrary. If for "can" we were to read *may*, this opinion might pass, but in a state so attenuated as not to be worth having. That a gradual privation of direct arterial blood, by silting up of the coronary or any other arteries, or stopping their mouths, is a painless process, is displayed to us every day. Yet physicians seem satisfied with the explanation, as were two eminent foreign physicians with whom recently I was in conversation. I reminded them that, as I have twice observed, hæmorrhage into the pericardium from a ruptured coronary was quite painless. It is the return of blood into anæmiated parts which may cause intense pain, not the anæmia. Why in some cases, even of *sudden coronary thrombosis*, there is pain, and not in others, is a problem which I shall attack presently (p. 450); rate and quantity seem to be the determinants. In some of these cases, as Sternberg[4] in a very interesting paper has shown (*vide* p. 461), the pain is associated with a patch of pericarditis, apparently produced in the same or a similar way as the patch of pleurisy over a pulmonary embolism.

Josué, in discussing cardiac aneurysm from the point of view of my hypothesis of angina, at which he had in-

[1] Burns, Allan, *Obs. on Dis. of the Heart.* Ed. 1809 (not in the libraries of Camb. Univ. or of Roy. soc. Med.). Kindly lent to me by Sir W. Osler.

[2] Charcot, *Gaz. méd. de Paris*, 1859, p. 283.

[3] Potain, *Dict. des sci. méd.*, Paris, 1866, sér. i. tome iv. p. 347.

[4] Sternberg, "Pericarditis epistenocardiaca," *Wiener med. Wochenschr.*, 1910, No. 1.

dependently arrived and has recently expressed almost in my words, says that in cardiac aneurysm pain is far from obligatory, and from most cases has been absent. With Paillard [1] he closely examined a certain case, in which the right coronary branch and all the collateral branches to the aneurysmal part had been blocked; but the patient had never suffered from any anginiform symptoms; and his systolic pressure was 180. Except about the aneurysmal area, where of course it had undergone the usual necrotic changes, the myocardium was normal, and indeed hypertrophied, because of a pericardial synechia, and also, as it was supposed, of adrenal hypertrophy (see Vol. I. p. 225). There was some atheroma of the aorta here and there, though this vessel generally was fairly supple. The kidneys were normal.

Von Romberg, with some confidence, but without producing much evidence beyond treacherous analogies, repeats the suggestion of Virchow, that the cause of angina may lie in embolisms of the coronary arteries, or of their branches, and compares the pain with that of sudden embolism of an artery of a limb. Death in senile angina may occasionally be due to a block in a coronary artery or branch, but it is difficult to suppose that for every attack there is a new embolus; and in embolism of a limb two pains are to be observed, the pain of the striking plug, due to lateral tension above it, which may be very sudden and severe, and the later continuous pain of acute obliterative arteritis; a disease not very comparable in any direction with the symptoms or the histology of angina. Moreover, what we know about thrombosis or embolism of the cardiac arteries, whether experimental or pathological, is not consistent with the sum of our experience of the conditions of angina, for of these blocks an immediate and considerable fall of blood pressure is, and must be, a character; yet during attacks of angina pressures are not infrequently high, and a fall of pressure, even by heart failure, usually abates it. Kauffmann, in the sixth edition of his *Lehrbuch* (i. 37), after commenting on angina as a symptom of coronary embolism, quotes a case in a man æt. 35. The symptoms were sudden agonising *dyspnea, fall of blood pressure,* and pulmonary œdema. Death in seven hours. There was a fresh plug in the left coronary artery; no other

[1] *Bull. et mém. de la Soc. Méd. des Hôpitaux de Paris,* 29 janvier 1909.

cardiac disease. I shall describe angina as a result of such an accident, but this case, so far as the history goes, was not one of angina; there was no pain, and dyspnea and asystole are not angina. As regards embolism in particular, this in the coronary vessels is probably a very rare event. Experimental embolism, even the finest, has been shown over and over again, recently again by Wassiliewski [1] with lycopodium seed, to cause a transient fall of pressure and of cardiac energy, a dilatation of the cavities, a diminution of the systolic wave, and a retardation of the beats; with a larger embolus these symptoms are not transient, but advance with the structural demolition. Such, *whether by block or spasm*, must be the instant effect of a sudden check to the coronary circulation. But is this what we see in angina pectoris? Surely not!

Von Basch's cases, if indeed all of them were angina, or anginous, are rather vague. As to the nature of some of them he himself was in doubt; others, if angina at all, were complicated with heart disease.

Many years ago von Leyden also,[2] after a careful survey of his materials, reiterated that angina pectoris was not only, as we all admit, often absent in disease of the coronaries, and also in fibrous degeneration of the cardiac muscle, but absent likewise in some cases of acute thrombosis, or infarct, of either of them, and in various combinations of such conditions.

Ligation has contributed its part to our knowledge of this subject. On tying a fairly large branch of a coronary artery, the systemic pressure falls at once, and the output also; then the pressure rises on the venous side; then beats drop out, and so on. Erichsen [3] clearly demonstrated this series of events in dogs and rabbits; and twenty years later Panum, in a better-known paper,[4] added little to Erichsen's demonstration. Von Basch and his pupils tied coronary arteries and twigs of them, and found likewise that the arterial pressure fell, the pulse became irregular, and pressure rose in the left auricle and lungs with œdema and death. To tie only one large branch sufficed to cause arrhythmia and death.

[1] Wassiliewski, *Zeitschr. f. exp. Path. u. Ther.* Bd. ix. H. 1.
[2] V. Leyden, "Sklerose d. Kranzarterien," *Zeitschr. f. klin. Med.*, 1884.
[3] Erichsen, *London Med. Gaz.*, 1842.
[4] Panum, *Virchow's Arch.* Bd. xxiii.

Porter's [1] researches on these lines are well known, and his results supported those of von Basch. He found that the time of the heart's arrest was, *caeteris paribus*, proportional to the size of the branch occluded. If either main vessel was occluded both ventricles stopped together. These experiments show of course the effects of sudden, not of gradual, occlusion. The very careful series of experiments on dogs and monkeys by Hirsch and Spalteholz [2] showed that by careful ligation of the descending branch of the left coronary, the initial arrhythmia (at the moment of ligation) might be transient, and the animals could be kept alive, though with evidence of greatly reduced cardiac potential, for three or four weeks, till killed for autopsy. Thromboses and callus were then found, especially towards the apex. To the careful injections by which they verified the fact that coronary anastomoses were free and abundant, being very evident in the papillary muscles, I have already referred. Dr. Parkes Weber found that when one coronary had been closed at a certain spot for a sufficient time, the channel beyond the block being patent, an injection into it soon flowed out at the other vessel. He showed also, as Professor Spalteholz had kindly demonstrated to me, that the interventricular branch of the left vessel on reaching the apex could be seen to anastomose with a twig of the right vessel. In conversation with Professor Spalteholz on the lymph channels he told me that the blood and lymph channels in the heart are so abundant and inosculate so freely, that during life the myocardium may be compared with a sponge. On such facts Sir William Osler may well comment: "These experiments, proving arrhythmia and arrest in diastole, do not throw much light upon the etiology of angina pectoris." He adds that even in cases of sudden death due to blocking of one of the coronaries, or of a large branch such as the anterior, the seizure is usually painless; the converse of an ordinary attack of angina in which pain is eminent and blood pressure, usually at any rate, maintained. Dr. Rolleston has published evidence to the same effect.[3] Huchard admitted

[1] Porter, *Amer. Journ. Exp. Med.*, 1898.

[2] Hirsch u. Spalteholz, *Deutsche med. Wochenschr.*, 1907, No. 20; with beautiful plates. See, to same effect, Porter and others.

[3] Rolleston, H. D., *Brit. Med. Journ.*, Nov. 1896.

that angina is not characteristic of, and rarely occurs in, cardiac aneurysm of coronary origin.[1] However, as we shall see in Sternberg's cases, angina may arise in this condition if pericarditis intervene; or even without it (p. 462). Huchard explains the absence of angina by the deadness of the portion of myocardium concerned; but a period, however brief, of acute ischæmia, with secondary congestion, must precede the necrosis.

In respect of this part of the argument: if it be contended that while in chronic cases coronary ischæmia may be too slow to set up angina pectoris, but that a sudden blanching might have a different and more intense effect from a slow anæmia of the myocardium, let us then consider the effects of a sudden ligation of a large coronary branch in an animal. We find that in 60 to 90 seconds diastole is extended, and pressures are falling; in 30 to 60 more seconds the carotid pressure runs down by 50 per cent, the heart loses its rhythm, and in a quarter of an hour ceases to beat. And so in man also a sudden plugging of a large coronary branch presents a like series of events. But are these symptoms, with the dyspnea which accompanies them, characteristic of angina pectoris? Certainly not. During an ordinary attack of angina the aortic pressure rarely falls; the heart does not characteristically lose its rhythm; it does not dilate; there is no dyspnea. Nay rather, have we not seen on the contrary that vasodilatation, or mitral regurgitation, factors of falling aortic tension, so far from calling forth or aggravating these alleged anginal effects of a failing coronary supply, generally counteract them. If by others it be urged that, granting such more or less rapid occlusions as are possible in experiment may produce symptoms unlike to, or even somewhat exclusive of, the phenomena of angina, yet we cannot be sure that a more gradual experimental occlusion might not produce different phenomena, it may still be demurred, not only that the symptoms of experimental occlusion are unlike angina pectoris, but that they dissemble any such kinship with remarkable uniformity. Even in a gradual occlusion we find on the whole a gradual reduction of blood

[1] *E.g.* see carefully analysed cases by Josué, *Bull. et mém. de la Soc. Méd. des Hôp. de Paris*; Séance, 29 janvier 1909.

pressures, cardiac dilatation, and an ingravescent tendency to syncope; but none of these acute and violent epiphenomena—variations surely not of quantity only but of kind.

Notwithstanding, I have admitted that in some of these cases of coronary thrombosis an intense anginiform pain may set in; these cases I am to consider later (p. 450); but here I must dwell a little longer upon the preliminary postulate—that in angina pectoris the coronary arteries are always diseased, or, at any rate, pertinently affected; a postulate which Merklen audaciously calls "Huchard's law." In weighing our data in this matter, we lie under the difficulty that defects of these vessels are conspicuous in the mortal cases, while in those which end in recovery—and they are not a few—there is no necropsy. Still, so far as I am aware, there is not a case of angina pectoris on record, on responsible authority, in which disease of the coronaries was the sole lesion; none, that is, in which, for instance, the state of the suprasigmoid aorta also had been histologically examined, within and without, and declared normal. Barié [1] admits that cases of angina without coronary disease are not very rare. Yet Dieulafoy, after recognising angina as arising from acute aortitis, in his section on this disease, when, in his chapter on Angina Pectoris he finds himself under the spell of the coronary convention, cannot but drop into the old chant—coronary disease, ischæmia of the heart, angina pectoris, death. Even Merklen (*loc. cit.*) had to admit that in angina the coronary arteries are not always diseased, when, losing his customary bearings, he strayed into vague conjectures.

Statistics taken upon records in the gross, on account of the imperfection of the data and the laxity of terminology, are so fallacious that we can place no great reliance upon them. I have made no such collection, and trusted no such inferences. Still, the computations of others need not be ignored; they have at least an auxiliary value. Dr. Graham Steell [2] concludes: "The disease (angina) may be met with apart from any lesion of the coronaries." Osler (*Lumleian Lects. loc. cit.*) in 17 cases—cases probably well verified—reported coronary disease in 13 only. Dr. Verdon, in a private letter, very kindly referred me to longer

[1] *Maladies du cœur*, 1912, p. 960.
[2] Steell, G., *Index of Treatment*, 6th ed., 1911, p. 67.

series ;[1] on a summary of 283 cases collected by himself, he found that of fatal cases of angina the coronary arteries were healthy in 43 per cent. Tacchi in 70 cases found coronary disease in 38 only ; Sir John Forbes in only 16 of 45 ; Lusani in 21 of 36. Laennec's experience was curiously negative. " I have opened," he says,[2] " several (plusieurs) subjects of . . . angina pectoris, but in none have I found (chez aucun je n'ai trouvé) the coronary arteries ossified." (The word " ossifiés " in the writers of the period included atheroma.) When we consider that angina pectoris is in large part a disease of advanced life, and of subjects of cardio-arterial disease, this low proportion does not remove our doubts of the validity of calculations on reports taken in the gross. Morgan,[3] in surveying the literature of angina, says that whereas coronary disease is found in many cases, yet " true " angina may occur in patients who are entirely free from disease of the heart. Such cases are more or less reluctantly admitted, even by orthodox champions, to be authentic.

Other cases are mentioned incidentally in this essay ; *e.g.* Heberden's (Comm. 296-7) case of sudden death in angina, of which he reports : " A very skilful anatomist could discover no fault in the heart, in the valves, in the arteries or neighbouring veins, excepting some rudiments of ossification in the aorta." Desportes likewise described a case of rheumatic aortitis and angina pectoris, in a man aged 25, in which the coronary arteries and their orifices were perfectly normal. By way then of concrete illustration only, I will cite here a few more examples of angina pectoris (mortal) without coronary disease.

Case I.—Baccelli's Clinic:[4] male, æt. 50; active and healthy, but a drunkard. Pulse 80, rather tense (no pressure record). No heart nor anginal symptoms till sudden attack of typical character, very severe. Pulse 100-120, tense (" teso "). Morphia injected, but the operation scarcely finished when the patient turned up his eyes and drew some deep breaths ; the pulse failed, and the heart ceased to

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[1] Verdon, W., *Brit. Med. Journ.*, March 18, 1911. See also Whittaker, *Twentieth Century Pract. Med.* vol. iv. pp. 439-441.

[2] Laennec, *Traité d'auscultation méd.*, 1831, vol. iii. p. 351.

[3] Morgan, *Journ. of Amer. Med. Assoc.*, July 1897.

[4] Baccelli, *Rep. Galli. ; Gaz. d. osped.*, Feb. 2, 1908.

beat; another gasp and life passed. P.M. heart "completely normal—valves, etc. Coronaries elastic and pervious (*elastici, pervie*)." No embolism. "Other viscera normal." (*No mention of the aorta*, nor note as to syphilis.)

Case II. (Merklen.[1])—Male, æt. 22. First seen in 1876 for aortic disease (very big ventricle). Potain's diagnosis was syphilis. (Note the early age.—C. A.) After a quiescent period, seen again at æt. 33: pleuritic and pericardial symptoms had appeared (syphilis about mediastinum?). "Plusieurs crises angineuses" and death. P.M. Aortic valvular disease, as above; and also *a very limited fresh lesion of the aorta forming a zone above the valve, and stopping close to it.* There was only one coronary artery, but this was normal throughout: many sections of its course were made.

Case III. (Jecce.[2])—Male, æt. 30: in hospital for angina pectoris; usual symptoms; died in an attack. Aortic murmur, direct and regurgitant. Syphilis; infection nine years before. (Never rheumatism.) Aortic cusps thickened; two adherent, causing stenosis. *Coronary arteries both healthy. Aorta "atheromatous."* (For atheroma read syphilitic aortitis.—C. A.)

Case IV.—One of Dr. Lindsay Stevens' cases recently published in the *Glasgow Hospital Reports* (no date on my offprint). Male, æt. 44; no doubt a true case of angina (many of the cases in his interesting lists could not properly be so called, and others were complicated with cardiac symptoms, such as dyspnea, etc.). After death there was "atheroma" of the arch of the aorta [probably at æt. 44 an infectious aortitis?], but the coronary arteries were healthy. The dilated and hypertrophied cardiac muscle had undergone fibroid degeneration.

Cases V., VI.—Our lamented colleague Professor Dieulafoy, in the fifteenth edition of his text-book, in describing multiple aneurysm of the aorta in *two* fatal cases of angina pectoris, concludes: "They succumbed, although there was no coronaritis; a proof that *obliteration of the coronaries is by no means the sole cause of angina pectoris*" (author's italics).

Case VII.—Dr. Mackenzie has very carefully described a severe case.[3] Male, æt. 35, in which however after death "wonderfully little change was found in these vessels." The right coronary, if somewhat small, was "apparently normal"; the left, "patent and large"—"perhaps slightly thickened, but not markedly so." Aortic regurgitation and enormous heart. "Aorta somewhat thickened by patches of commencing 'atheroma'" (syphilitic aortitis?). There was some fibroid deterioration of the myocardium.

[1] Merklen, *Troubles fonctionnelles du cœur*, 1908, p. 162.
[2] Jecce, *La Rif. med.*, April 2, 1897.
[3] Mackenzie, Jas., *Heart*, vol. ii., 1911.

Case VIII.—In the chapter on Aortitis (p. 158), and again in this essay (p. 275), I have quoted Dr. Poynton's case of angina in rheumatic fever, diagnosed by Cheadle as "true angina"; in this case the coronaries, including their finest ramifications, were healthy.[1] "*Acute aortitis only was found to account for it*" (italics mine). (See also pp. 435 and 447.)

Case IX.—Dr. Poynton quotes another case to the same effect (see p. 275), and comments that "although one would naturally guess, when sudden death with or without angina had occurred, that the lesion was so distributed as to have closed the coronary orifices, yet that in the two cases here recorded I do not think that such an explanation is tenable."

Cases X., XI.—Dr. Raw[2] describes two cases of angina with disease of the aorta, but "the coronary arteries in both these were patent, and there was very little evidence of (general) arterial degeneration."

Case XII.—Reported by Andrew Clark[3] (from the London Hospital). Angina pectoris, chest and left arm; aggravated by effort. Blood pressure high; radials thick. P.M. aortic valve competent, but "somewhat" diseased. Hypertrophied left ventricle. First part of aorta much diseased, but beyond arch healthy. "*No disease of the coronary arteries, and nothing found wrong in the heart to account for the symptoms during life*" (italics mine). "Syphilitic gummata" found in the liver. No other sign of syphilis. (No doubt a syphilitic aorta.—C. A.)

Case XIII. (related by Ortner.[4])—The history was of attacks of angina in which pericarditis (*vide* p. 454) had also been diagnosed. But at the necropsy was found not the serofibrinous kind of pericarditis expected, but an advanced sclerotic process involving the ascending aorta and the arch, with many "*atheromatous ulcers, one of which had penetrated through the whole thickness of the wall*" (italics mine). There was thus a "periaortitis at the root of the aorta, which had extended to the neighbouring pericardium." Now the only coronary affection was advanced change (*hochgradige Veränderung*) about the orifice (*Ausgang*) of the *left vessel only* (to which of course Ortner attributes the angina). In no other wise were these arteries affected; nor was there any consecutive softening (*keine Myomalakie*) of the myocardium. No great harm then was done to the heart by the partial blocking of one of the orifices. The case was probably one of syphilis.[5]

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[1] Poynton, *Lancet*, May 20, 1899.
[2] Raw, N., *Lancet*, Aug. 21, 1909.
[3] Clark, A., *Lancet*, Aug. 31, 1878.
[4] *Loc. cit.* p. 13.
[5] See also for additional instances, Morel-Lavallée, "L'Angor pectoris non-coronarienne," *Rev. de méd.*, 10 oct., 1899.

Case XIV.—A similar case, most carefully described by Rist and Krantz.[1] Did this case stand alone, it would be conclusive against the obligate association of angina with coronary disease (see p. 195).

Case XV.—Dr. Hirschfelder most kindly sent me the second edition of his important work on "Heart Diseases," but after these pages were in print. I had just time however to quote from him a case at the Johns Hopkins Hospital, five years of suffering from angina pectoris. An aortic cusp was ruptured; the coronary arteries were soft, and their walls everywhere normal. The aorta is not mentioned.

To proceed more succinctly: Hoffmann of Düsseldorf[2] says he has "often observed, . . . after years of the severest attacks of angina pectoris," that the necropsy did not show the slightest change (nicht die geringsten Veränderungen) in the coronary arteries. Of five of Latham's necropsies of angina, three presented coronary atheroma, two "fatty degeneration of the myocardium" only; of these two Arnold's was one. (Arnold's heart possessed but one coronary artery, which was not diseased.) In a case of syphilitic angina (periaortitis and aortitis), published by Dr. Morison,[3] the coronary arteries, though in an abnormal position and somewhat small, were not diseased. My Oxford brother[4] quotes with emphasis, one after another, three cases of typical angina (syphilitic aortitis?), in each of which the coronary arteries and their orifices were unaffected. He does not draw the conclusion that coronary disease is not essential to angina pectoris, yet is not this conclusion—to use Vaughan's word—inexcussible? Hirschfelder's case[5] from the Johns Hopkins Hospital I have quoted already; Von Leyden indeed, in the conclusion of his essay (*loc. cit.* p. 559), admits that angina may occur without any coronary disease, and especially cites cases of syphilis, and some other infections, to this effect. (See also case p. 458.)

Dr. Stengel,[6] in the course of an interesting paper on the nervous symptoms of arteriosclerosis (see essay on Arteriosclerosis) writes: "In sclerosis of the arch of the aorta painful

[1] Rist and Krantz, *Bull. et mém. de la Soc. Méd. des Hôp. de Paris*, 28 juin, 1906, p. 653.
[2] Hoffmann, *Diag. u. Therap. d. Herzens u. Gefasse*, 1911, p. 334.
[3] Morison, A., *Lancet*, Feb. 3, 1912.
[4] Osler, Sir W., *Lumleian Lect.*, p. 839-840.
[5] Hirschfelder, *Diseases of Heart, etc.*, 2nd edition, p. 376.
[6] Stengel, *Amer. Journ. Med. Sci.*, Feb. 1908.

paroxysms occur on slight exertion, which may have the character of angina pectoris, *even though the coronary vessels are not involved*" (italics mine). Cohnheim[1] says, as to the "causal relationship of angina with coronary disease, . . . often there is no trace of disease to be found in the coronary arteries of such subjects, and what is still more common, very high degrees of coronary sclerosis are met with in persons who have never had an attack of angina." It is true he tried to whittle down the distinction rather fancifully; *e.g.* that aortic pain is more "diffuse," and is in closer relation to effort than in angina from disease of the coronary vessels; but he shifts the *onus probandi* upon the coronarians.

Again, if it be agreed that "abdominal angina" may arise from disease of this portion of the aorta (*vide* p. 309), what need is there to drag in the coronary vessels? As regards fibrosis of the heart (considered in section on Cardiosclerosis (p. 21), alleged to depend on coronary disease, and often associated with it, we know that the presence of these nodules and diffuse fibrous hyperplasia does not produce angina; nor indeed any definite derangement of the circulation, unless perchance a heart block. That in gradual sterile thrombosis of coronary branches, of not more than middle magnitudes, no important symptoms of heart failure (after the initial symptoms) ensue, although callus and fibrosis may be found after death, was Kanthack's conclusion; and it is strongly supported by Krehl,[2] who had often found extensive change of the kind without record of impairment of cardiac function during life, and by many later pathologists. Yet surely in no condition would the alleged "partial myocardial cramps," or "fractional systoles," be more likely to occur. Anginous symptoms on plugging of a main branch, or ramus descendens and circumflexus, with acute myomalakia, will be considered presently.

Such is the testimony of no very favourable witnesses to the not infrequent absence of coronary disease, even in mortal cases of angina; surely then in the cases which by recovery elude our proofs, we who from indirect evidence infer coronary integrity are not without justification. Although for years I have insisted on this happy issue of many cases, I have been till lately alone

[1] Cohnheim, *Syd. Soc.* ed. 1889, vol. i. p. 37.
[2] Krehl, *Lehrb. spec. Path.*, 1902.

in recording cases of recovery. Perilous as the malady is, death is not of its essence. I shall declare that defect of the coronary arteries, together with other causes of myocardial decay, does indeed take a large share, not in the generation of angina pectoris, but in the mortal issue of it.

I repeat that I am no longer alone in recording cases of recovery; Sir William Osler has reported such cases. But, our critics retort, "then they were not angina pectoris"; all cases which do not fit their formula are to be discarded. If the patient died, the case was angina; if he did not, identical as the symptoms might have been, well, it was not angina. In other words, I am to have all the points I demand, or I won't play. Of such a contested case, Dr. Samuel West says (see p. 271) that "it had the same characters as angina pectoris"; in other words, a physician of his ability and experience could see no clinical difference. How can we then insist that the disease was not itself, but something else in the same form? Is this, in Professor Bateson's words, "to treasure our exceptions"? A more effective reply would be that, recovery or no recovery, the coronary arteries are always diseased, but not always so far as to forbid resumption of function, at any rate for a time, with remission of the symptoms. And in some elderly persons, in whom angina appears, disappears, and reappears, such may indeed be the explanation. Dr. Morison has written, and rather surprised me when he wrote[1]: "I am not aware of any record of the recovery of any case of coronary angina, though slow in development and compatible with a long life"; for even if by "coronary angina" Dr. Morison means cases of senile angina in which coronary disease may be presumed, even from my own small circle of experience I can adduce four or five such cases at least. And this explanation will not by any means cover those cases, not infrequent in younger persons, in which angina arises with great severity, and disappears—as, indeed, in them it usually does disappear—without ultimately any trace of harm, so far as the angina was concerned. Here let us remember again Broadbent's gibe on those who seek for recoveries in the post-mortem room.

That disease of the coronary arteries does not of itself set up angina, is an axiom founded upon universal experience. These

[1] Morison, A., *Lancet*, 1902.

vessels are atheromatous in at least three-quarters of the bodies which are found after death to be atheromatous at all [1]; indeed few elderly persons die without as much coronary disease as would content a coronarian disputant, yet angina is one of the rarer diseases. Potain argued that in many of these cases the coronaries though calcified might be pervious; and if so there would be no angina. Well, this is one good step forward. It is true that even in tortuous and knotty vessels the epithelial mosaic, which preserves them more or less from thrombosis, is very persistent, except in syphilis, in which infection, as I showed, and Dr. Aldren Wright verified, thrombosis occurs much sooner. Then what is the factor *x*—Thorel's "unerklärliches Etwas"?

What is the alleged mechanism, whether in the coronary area or not, by which this flare occasionally breaks out? The chief conjectures are four, two of them mutually contradictory; namely, first, that the occasional factor is a cramp of the cardiac muscle; secondly, that this muscle is painfully stretched; thirdly, that the left ventricle wrestles against aortic pressure; the fourth is the pretty fancy of intermittent claudication of the heart, which of late years has obtained many votes, and even the suffrages of Sir William Osler and Dr. Parkes Weber.

When we come to reflect, so far as our knowledge goes, upon the main facts of this part of the subject we may find it difficult to be serious in a discussion in which the part of coronary disease in the causation of angina pectoris is inculcated with almost theological impatience while its champions are content to attach the disease to coronary causes by links so slender, and to interpret it by arguments that are, more than one of them, mutually destructive. As Dr. Knott has remarked, "It requires no very profound knowledge of logic to see that a chain of which three essential links are subject to instantaneous evaporation, has ceased to function as a bond of union between fact and theory." But it would appear as if soundness on the coronary question may liberate the disciple to say what he pleases about other parts of the problem.

[1] See a large collection made in the Hospital at Ivry by Auscher and Pilliet, *Soc. Anat.*, Paris, October 2, 1891.

As with the advocate—it was not Taffy who stole the shin of beef; or, if it was, then in the alternative nobody saw him do it; or, if some one did, then the beef did not belong to the plaintiff: so of angina we are told—that it is due to coronary disease, because this defect causes cardiac cramp; or, if cardiac cramp cannot be, then in the alternative, cardiac dilatation; or, if cardiac dilatation is inadequate, then cardiac claudication; or, if in angina the heart does not "claudicate," then the case was not angina pectoris at all! By these sophistications we have seen one well-known teacher driven to the amazing assertion that "the intensity of the anginous symptoms corresponds with the intensity of the heart disease"; while another says magisterially, "There is a general consensus of opinion that those heart affections are most characterised by pain which at autopsy present most evidence of cardiac degeneration." Could the tyranny of notions over experience go farther than this? More open-minded physicians have found themselves compelled to admit that angina the most intense is, like aneurysm, compatible with a heart substantially sound, or even in normal condition; while, conversely, the stealthy way of cardiac degeneration is but rarely enlivened by interludes so dramatic.

Von Romberg, a devoted adherent of the coronary cause, says, in the new edition of his work, "a close observation of cases without heart weakness will prove to us that a reduction of heart energy cannot be unconditionally attributed to the conception (*Bilde*) of angina pectoris." It is strange that he does not see that, if so, the bottom has fallen out of the cardiac idea of angina. And these objections cannot be made of none effect by wilfully pretending that if the causes are not such as we have postulated then angina is not angina. Heberden said plainly that the cause is not cardiac, because he had noted *pulsum naturalem ut plurimum*; and are we now, on the rebuff of the necropsy, to protest that, although every term of the morbid series may be present in typical form, yet unless and until it is agreed to attribute to the coronary arteries the chief place in the causation we will plead an alibi or an alias, the name of angina shall be withheld at pleasure? Call no man anginous till he is dead; and not even then unless he has played the game! Thus it is that, with the modern development of neurology, the "soft options"

of neurosis, neuralgia, and so forth, return to favour; and therewith a wandering after any view which in the haze seems vaguely attractive. Now let us take the four guesses in turn.

Cramp of the Heart.—The hypothesis that angina pectoris consists in a cramp of the heart has been, not unnaturally, a prevalent speculation; for by no extravagant metaphor the anginal grip may be called a cramp. Heberden, although he regarded the heart as outside the argument, thought there was a "cramp somewhere"; and among other reasons dwelt on the nocturnal attacks, and on the free intervals, as in epilepsy and asthma. We have seen that Erasmus Darwin [1] and later physicians postulated a spasm of the diaphragm (or gout of this muscle), and of other thoracic muscles, a notion recalled by James Mackenzie. M'Bride, Baumés, von Dusch, Latham [2] and Walshe vulgarised this conjecture of a spasm of the heart, due in Baumés' opinion to foul blood. But upon cramp of the heart as a whole I need not spend much time. In my earliest papers I insisted that, certain poisons apart, the heart does not pass into a prolonged state of contraction, a statement which rests upon clear physiological doctrine; and now on clinical grounds Dr. Mackenzie confirms the opinion. Immediately on contraction the function of contractility falls into abeyance. Automatism, which in skeletal muscle is artificial, in the myocardium is a natural state. Calcium, which is necessary to the myocardium, to voluntary muscle is inhibitory. The chemical and electrical conditions are not identical. Cramp, or clonus, is not the abnormal but the normal action of this unique muscle; an action not balanced by antagonists, nor delivered by parcels. Besides, under our very eyes during the anginal seizure the heart does not stand still in systole; in most cases it continues normally to expand and contract. As Wassiliewski remarks (*loc. cit.*), spasm of the heart in angina is impossible if the cause be any kind of coronary closure, for this event is attended not with constriction but with dilatation of the heart. Von Dusch, too clear-sighted to ignore all this, explains that by "cramp" he meant something else, namely, "kurze rasch sich folgende Contractionen," but he does not explain how

[1] Darwin, Erasmus, *Zoonomia*, vol. iv.
[2] I think in his paper in *Med. Trans.* vol. iv. p. 278.

with a normal rhythm and output this can be. But it has been urged, as by M'Bride for instance, that after death the ventricle is found tightly contracted. Now it is on the diastolic phase, and on the later part of it, that the vagus tells. Well, for my part, poisons, such as certain glucosides, or sodium salts (Gaskell) excepted, I disbelieve in " deaths in systole " ; in systole the egg is on end. Post-mortem systole is usually rigor mortis. We can no longer suppose that the form of the ventricles after death, a form usually accidental, throws any light upon the condition of the heart during life. Balfour says definitely (*loc. cit.* p. 316) that in death from angina the left ventricle may be either contracted and empty, or flabby and full of blood ; I can testify that the ventricles may be relaxed and full of clot. Mackenzie remarks that in angina by a summation of stimuli the heart becomes painful, but what may be the attitude of its muscle during the attacks he does not tell us. However the alleged " cramp " of the heart, or its parts, he admits to be unthinkable. Sir James Goodhart points out the difficulty, which should be obvious, of supposing such a cramp to have no effect upon the pulse. Dr. Morison [1] supposes a " crushing in its tonic grip the sensory nerve endings of the organ." What and where ? In his histological introduction he gives no description within the muscle of *afferent* end organs (see p. 411). And what about the crushing of any such end organs in ordinary systole, or in the systoles of renal and other vehement hearts ?

Yet while heart cramp is thus driven from pillar to post, its supporters still cling to fragments of it, and declare for " localised (partial ?) cramps," as first suggested by Kronecker. By " localised" cramp is meant, as I am informed, a contraction in a segment or segments of the myocardium prolonged beyond the cyclic systole. It is not easy to picture a cubic inch of the ventricular wall, woven as it is, being held in persistent contraction, while the portions on all sides of it, as the pulses tell us, are pursuing their rhythmical periods and discharging their functions adequately ; nor again to explain how such a fragment, if abutting upon oscillating parts, could persist in spasm without upsetting the balance of the whole. We might try to fancy a

[1] Morison, A., *Cardiac Pain*, p. 277.

sector of the muscle, in form like those natural divisions of an orange which children call "pigs," as held in a discordant tension; but when we come to think of the fibres of the muscle, not thus departmental but crossing each other's lines of force in many planes, we realise that this fancy must be unsubstantial. And fractional systolic efforts of the kind supposed should be visible in the electro-cardiogram. The cardiac muscle, whose fibres are not insulated by sarcolemma, does not, after the manner of voluntary muscle, sustain a contraction by alternations of fractional contractions, but by one consentaneous and maximal effort of all its integrated fibres; an effort which must be followed by a like universal relaxation. Of course if by coronary embolism a parcel of the myocardium softens, a partial diastole necessarily ensues; and generally speaking, the effect of malnutrition is a blocking or delay of the contractions. Were we loosely to compare fibrillation of the auricle with "partial spasm," fibrillation does signify suspense of the function of the whole chamber. As Dr. Rolleston [1] has said, the only conceivable seat of partial spasm is in the musculi papillares; but, were there no other objections, it would be sufficient for the dismissal of this alternative to remember the maxim that, in respect of the parts of the heart, angina is associated with the aortic; with the mitral part very rarely. Well then, replies one able and pertinacious disputant, "the heart muscle, if not capable of spasm, may be capable of an abnormal contraction akin to spasm"—a dilemma worthy of Duns Scotus, a distinction which may be left to die of its own attenuation.

Dr. James Mackenzie attributes the "gripping sensation of angina," to "*spasm of the intercostal muscles*," and so decisively that he "would even limit the name to cases in which, besides the pain, there is a sense of contraction in the chest . . . as if the breastbone would break." [2] In one of his published cases Dr. Mackenzie graphically describes an experience only too common in angina pectoris, that "he (the patient) dare not move for fear of the awful pain . . . a terror such that sweat poured off him"; but this stillness he does not attribute to the dread or agony, but, on Erasmus Darwin's hypothesis, to the cause of

[1] Rolleston, H. D., *Med. Soc. of London*, Feb. 24, 1902.
[2] Mackenzie, J., *Dis. of Heart*, p. 38.

the agony, a spasm of the muscles of the chest,[1] to which however Darwin added spasm of the diaphragm. A similar explanation was advanced by Baumés, who, however, attributed a part of the fixation of the chest to ossification of the ribs. Now Wichmann observed the thoracic muscles carefully, and concluded that, although there is a sense of constriction, the thoracic muscles are not contracted—the respiration is overawed, but not locked up. Jurine says that a deep sigh may be drawn, and this I have seen more than once, even sighings. The respiration in angina is often abated or in abeyance, it is true, but, in my opinion, by vagus or other inhibition. Dr. Morison, in a large operation for synechia,[4] observed that post-sternal angina persisted notwithstanding the division of the 5th and 6th intercostal nerves, and removal of the intercostal muscles. Now the vice-like pain of angina—major and minor—although of all degrees, from "stenocardia" to unutterable agony, is in kind, in seat, and in menace, peculiar; it varies only in the number of the turns of the screw. The intercostal muscles, when in action, do not crush the breast-bone inwards; they expand the chest. Moreover, if in cramp, they would, as in all muscular cramps, give pain in proportion to their volume. Cramp of the foot is vexatious, of the leg it is distress, of the thigh it is the rack. The intercostal muscles are short and thin; they are many it is true, but even if unanimous have nothing like the volume to set up a pang so poignant as that of angina. A frequent area of anginous pain is that of the left pectoral muscles, but it is not by any constraint of theirs that the patient may be deterred from gripping the arm of his chair, nursing his left arm across his chest, or taking the medicine cup in his hand; he is transfixed by the writing on the wall. He could move an he dare. In muscular cramp we may howl or rage; angina pectoris is beyond all raging. By the bed of a strong man in an attack of the major angina the very chamber is silent with awe. In this agony—the uttermost torture, so we are told, in the rack of disease—the wit of the physician only is still vigilant; and in this strait can he see no more than the plucking of a few chords

[1] Dr. Mackenzie has suggested that the pain of pleurisy is, in like manner, due to intercostal spasm. Likewise in the older authors we find anginous symptoms attributed to ossification with rigidity of the ribs.

[2] Morison, A., *Lancet*, Jan. 8, 1910, p. 99.

of fiddle-string muscles at the patient's ribs! The anguish is of far grimmer, far deeper significance. Even of attacks of trigeminal neuralgia no man has died. Some variable reflex fixation of those muscles there may be; in diseases of the thoracic viscera, as Wolff-Eisner has demonstrated in phthisis,[1] the muscles of the walls are in spasm, and may be felt thus rigid; but angina is no consequence of this. Besides, the notion bears not at all on the pain of angina minor, which, as Dr. Mackenzie himself says, "may be but a slight sensation," yet the pain is in nature identical. In the angina of acute aortitis (as in Dr. Christian Simpson's case (p. 274) and others of status anginosus) I have had time and to spare for observation of the walls of the chest, and am in a position to assert that, in this acute form of the disease, no such locking of the intercostal muscles is to be discovered. Moreover the pang is usually substernal about the manubrial joint, away from the intercostals; and the grip is described as inward, not as a stitch but as crushing by a heavy bar, or a vice-like compression of the breast towards the spine. Furthermore in many anginous patients the lungs are emphysematous, and the ribs rigid already. As I have said, the older writers, such as Wall, with some consent emphasise ossification of the ribs as a feature, if not a factor, of the disease.

Distension of the Left Ventricle. — Now let us pass on to the notion of ventricular dilatation—brusque distensions. Of this hypothesis the supporters are Potain, West, Merklen, Teissier, and many others. It seems odd in one breath to assert that angina pectoris is a state of high pressures, to be combated by nitrites, and yet that the ventricle is dilating; in this case, as Dr. Colbeck has argued, pressures should be falling. Of the distension alternative I cannot select a more weighty exponent than Dr. Samuel West, who with rather surprising readiness[2] remarks that "sudden dilatation of the heart produces angina pectoris, as our daily experience tells us." My "daily experience," and I venture to think the general experience, is that the heart is liable to be far more dilated, and more suddenly dilated, whether on the right

[1] See Pottinger, *Deutsche med. Wochenschr.*, April 21, 1910.
[2] West, Samuel, *Brit. Med. Journ.*, June 23, 1906.

side or on the left, in disorders and stresses other than angina, and this without any considerable pain; while conversely in angina dilatation of the heart, whether we look to the cardiac physical signs or to the secondary evidence of other organs, is no considerable feature. We often meet with extreme dilatation, as for instance in infective myocarditis, with no pain, although the sign of pain would be a useful signal; there is no approach to a parallel between cardiac dilatation and cardiac pain. And when we do find dilatation and pain together the pain is not characteristically anginal (pp. 430 and 441). Indeed, as we shall see presently, Dr. West, although he did emphasise dilatation, does not now exactly postulate, as the main factor, dilatation only. All the evidence on the effects of nitrites, of rest, of mitral regurgitation, and the like, goes to prove not that the heart is failing in tone, or contractive power, but, relatively to the angina, is doing not too little but too much. Even Huchard, not seeing how it told against his coronary hypothesis, admitted, beyond the facts: "L'angine de poitrine ne peut plus se produire chez un malade en état d'hypotension artérielle." I have already quoted (p. 331) Dr. Mackenzie, and many other authors, to the effect that during an attack no alteration of the heart's dimensions is to be detected. Merklen (*loc. cit.*) says bluntly that dilatation, as an explanation of anginal pain, will not do; "elle est, somme toute, chose banale." Moreover cardiac dilatation is apt to set up dyspnea, which is not a feature of angina. That, as Balfour, Broadbent,[1] and Musser (*loc. cit.* p. 389) pointed out, and as others, such as Sir Richard Powell,[2] Sir Thomas Oliver,[3] and Dr. J. S. M'Kendrick,[4] have verified, angina pectoris is relieved by mitral regurgitation, and, as Dr. Mackenzie [5] stated of "a number of cases," "ceases with the onset of auricular fibrillation," does not look as if dilatation of the heart were the cause of the pain. Nay rather, signs of yielding heart with its consequences, when the coronary blood pressures must be at their worst, are not infrequently signs of departing angina; and the action of the nitrites is not to

[1] Broadbent, W. H., *Lancet*, May 27, 1905.
[2] Powell, Sir R. D. See *Report of Harveian Soc. Discussion*, loc. cit.
[3] Oliver, Sir T., *Lancet*, Sept. 1905.
[4] M'Kendrick, J. S., *Glasgow Med. Journ.*, Nov. and Dec. 1909.
[5] Mackenzie, James, *Brit. Med. Journ.*, Oct. 31, 1911, p. 971.

withstand dilatation. By what mechanism does syncope or mitral regurgitation fill the coronary arteries, the emptiness of which is on current hypotheses a primary condition of the angina? It is generally agreed that the onset of angina may be accompanied not by falling but by rising pressures, whether primary or secondary; that indeed, though during enormous rises of arterial pressures intracardiac diastolic pressure may be constant,[1] the attacks are, almost as a rule, determined by momentary rises of intra-aortic pressures. We shall see presently indeed, that it is not intracardiac but intra-aortic tension which excites anginal attacks. One of the most sudden and severe modes of dilatation of the left ventricle which can happen is that ensuing upon a rupture of an aortic cusp; now, although the distress of this rupture is severe, unless there be such other lesion as to give rise to angina, the characters of this accident are not those of angina; the distress is different, more persistent and attended with dyspnea. As Ortner, in his elaborate report on Herzschmerz,[2] sums up this matter, "the pain of dilatation, if any, is a much smaller affair, causing an aching, or possibly a stitch, in the submammary area, and superficial cutaneous tenderness." For these and many other reasons mere distension, which Parry, supposing that it tended to "exhaustion of the heart's irritability," seems first to have assumed, which Fothergill and Stokes seem to have meant by the phrase "weak and fatty heart," which Traube interpreted as a dragging upon the nervous fibrils in the wall of the tauter ventricle, and which more than twenty-five years ago von Leyden[3] and other physicians, abroad and at home, still held to be the condition of angina, but which Vierordt, at the Wiesbaden Congress of 1891 demolished, has been dropped; although every now and then it is introduced furtively when the disputant is in straits. But, as Dr. Nathan Raw remarks,[4] no one has ventured to say that angina is relieved by the "re-establishment of compensation"; though Curschmann contrariwise made the curious remark, that with an extremer degree

[1] Hill and Barnard, *Anat.*, 1879.

[2] Ortner, *Jahreskurse f. ärztl. Fortbildung*, Feb. 1911.

[3] Von Leyden, *Zeitschr. f. klin. Med.* Bd. vii., 1884 (angina as a periodical asystole).

[4] Raw, N., *Lancet*, Aug. 21, 1909.

of the coronary disease and corresponding enfeeblement and degeneration of the myocardium, he had seen angina diminish and pass away; and Ortner is inclined to agree with him.[1]

Fiessenger,[2] who cannot get rid of the coronary artery decay and consequent ventricular "fatigue," admits that he is perplexed and cannot understand his own interpretation, "because the myocardium is insensitive, and ordinarily myocardial failure is painless." So he guesses around the cardiac plexus, etc. His classification of anginas is artificial, not based on the deeper affinities.

Let us now go a step farther and refer to a paragraph in the late Professor Musser's well-known article[3] in which he not only denied that dilatation was the cause of angina, but argued, on the contrary, that by cardiac dilatation or mitral regurgitation angina pectoris is relieved (see Head, p. 300). Musser cited the following three cases. The first was one of angina with high blood pressure, loud second aortic sound, and so forth; the state became worse, and dilatation ensued, whereupon the angina was not exasperated but ceased. The second case illustrated the same sequence of events; on "rapid dilatation the angina ceased." And so it was with the third case; but in this instance when by restorative methods the hypertrophy of the heart was re-established, then the angina reappeared. Balfour's leading case was even more striking. He happened to be listening at the heart of a patient when an attack of angina set in, and he found it possible to continue the observations throughout the attack. The pain was the ordinary crushing of the sternum to the back, and radiation into the left arm. The radial pulse was tense. Gradually a mitral regurgitant murmur made itself heard, when the pulmonary second sound became accentuated and booming, the pulse small and feeble, *and as "cardiac asthma" set in the angina ceased* (itals. mine). It cannot be urged in these and such cases that it was a fall of intraventricular tension on the left side, transferring the stretch from the left to the right ventricle, which alleviated the angina. Intraventricular pressure is a very complex subject; it is suffi-

[1] Ortner. See *Fortschr. d. d. Klinik*, ed. Klemperer, Bd. i., 1910, p. 338.

[2] Fiessenger, *Acad. d. Méd.*, Paris, Oct. 1, 1912; well reported in *Lancet* and *Brit. Med. Journ.* for Nov. 9, 1912.

[3] Musser, *Amer. Journ. Med. Sci.*, Sept. 1897.

cient here to point out that it was at the moment when, relative or positive, the pressure seems to have been excessive for that ventricle, at the moment when it was so stretched as to give way, when the hydrostatic pressure of the column of blood in the left auricle began to tell on every square unit of the yielding intraventricular surface, that the angina did not come on, but went off! Indeed the maxim of Musser and Broadbent that dilatation of the ventricle mitigates angina suffices almost alone to extinguish the notion that the pain is due to distension of the muscular tissues or drag upon the enclosed nerves.

Systolic Effort.—But now we have before us an amended pleading; and we are indebted again to Dr. West and Sir Lauder Brunton for the alternative proposition, that the pain of angina is caused not by the distension but by a recoil or counter-effort of the heart against a rising intraventricular or intra-aortic pressure; "contracting against resistance."[1] In other words, the amended plea is that the pain is due, not to passive stretching, but to effort of the muscle; but yet, in the opinions quoted, is generated in no investments or attachments, but in the muscle itself. This change of the point of view is so important that I must quote Sir Lauder Brunton and Dr. West textually. Dr. West[2] drew a parallel between the contraction of the heart upon its contents, and that of the bladder, intestine, or other hollow organ upon its contents, when the issue is obstructed; it is "a spasmodic contraction to get rid of its contents"; and accordingly he called angina "cardiac colic." In similar terms Sir Lauder Brunton said, "angina is the pain of a tight ventricle in its ineffectual attempts to empty itself against the excessive blood pressure in the aorta"; he too compares it to colic, as when the colon is urging against obstruction; or to pain in the bladder, as when the expulsion of the urine is hindered; or again to the effort of the uterus at the beginning of labour. Massay's article[3] is but a version of Brunton's; he brings in likewise the bladder analogy, and so on. Gibson said that this notion in angina of "strife to surmount an obstacle" was first suggested by Cahen,[4] but the same view of the matter was also expounded

[1] See *Belfast Discussion*, 1909, *Lancet*, Aug. 7, 1909.
[2] West, S., *Brit. Med. Journ.*, June 23, 1906.
[3] Massay, *Paris méd.*, 1912, vol. xxxii.
[4] Cahen, *Arch. gén. de méd.*, 1863.

in an able paper, published about the same time, by Eichwald [1]; until recently however it had found few adherents. All these authors have regarded the pain as due, not directly to the tensile stress on a passive ventricle, which hypothesis they implicitly demolish, but to a contrary effort—an effort they would compare with colicky contractions of the uterus upon a clot. The rather vague opinion of Bamberger, that angina is due to "over-action of the heart," may in substance be the same. Now let us consider this crampy dilatation; this stretched contraction or contracting stretch, analogous to the pain of a hollow muscular organ, such as the urinary bladder when distended by a content which at the same time it is striving ineffectually to expel—an illustration which, like Mr. Pickwick's dark lantern, bewilders without enlightening us. In the case of the distended bladder, the pain is probably not in the muscular coat, but is set up by the drag upon the parts about its neck and the prostate; the fine nerve-twigs passing to the fibres of visceral muscle are regarded by physiologists as motor. So again the pain of colic is due, not to the contraction in the muscle itself, but to its abnormal pull upon the mesentery, a subcutaneous structure (p. 421), largely provided with Pacinian corpuscles. It is to such a tension at the neck of the aorta that I attribute angina pectoris; but, however this may be, the bladder, in the case supposed, is striving with a content which is not expelled; the heart on the contrary in the attack of angina, even during the worst of the paroxysm, is continuing steadfastly to deliver its tale of blood, per beat and per minute, with a tranquillity and effect which under any hypothesis are rather astonishing. And this it does often against high pressures. But both in health and disease the heart is continually called upon for such efforts—in disease of other kinds it may do it perennially, yet in such frequent cases we hear little of angina major or minor; anginoid pain is no characteristic symptom of aortic stenosis, nor of chronic renal disease, nor of hyperpiesia, even under the extremest aortic pressures. Moreover in these conditions we find the left ventricle hypertrophied accordingly, and, in the analogous cases, the muscular coat of

[1] Eichwald, "Über das Wesen der Stenocardie," *Würzburger med. Zeitschr.*, 1863, S. 249.

the bladder or stomach ; but of angina such a hypertrophy is no ordinary feature. To conceive of the pain of angina as due to muscular recoil, or a wrestle against abnormal resistance, is surely to creep back very near to the notion of cramp, and to attribute to the striated cardiac muscle the mode of activity of the unstriped muscle of the intestine, the bladder, and the womb, and to forget that this labouring of the heart is not, as in the cases given as analogies, ineffectual, but effectual : it is discharging its contents equably as in health.

Again ; we are not told what the excessive resistance is. Certainly the intra-aortic pressure is by no means constantly raised, and to regard normal aortic pressures as relatively excessive, is to shift our ground and to return to cardiac incapacity. Shall we not hesitate then to compare the maximal rhythmical discharge of their contents by the ventricles, even if it be against a high aortic resistance, with the slow cumulative progressions of unstriped muscle against an undischarged content ? We know that every contraction of the heart is a maximal contraction, whatever the residual blood ; but in cases of angina we have no evidence that residual blood is larger than in other and far more frequent states of high pressure without angina.

If it be replied that in coronary disease the heart is sensitive in a way that it is not when the coronary circulation is unimpeded, we have to enquire how defective nutrition makes for a " tighter " or strenuously " recoiling " ventricle ? Besides, the suddenness of the seizures is not suggestive of an accumulating resistance against the heart's expulsive efforts : surely the climax should be accompanied or even preceded by some sign of cardiac embarrassment, such as palpitation, or arrhythmia, or some physical signs of stress. And if squeeze of its intrinsic nerve-ganglia by a wrestling myocardium were the cause of angina, should there not be some parallel between angina and hypertrophy ? Once more, were angina due to any particular attitude of the unhealthy or healthy ventricular muscle, if, for instance, on each laborious heave the intraventricular pressure were forcing outwards any softened parcel of its mass, the pain should surely coincide more or less with the cardiac beat.

Merklen, after quoting with approval Sir Lauder Brunton's

comparison of the heart in angina with the bladder striving against an obstacle, and likewise seeming to forget that a bladder which cannot empty itself is not comparable with a heart which can and does empty itself, admits nevertheless that he is puzzled. He also is wistful for the "unerklärliches Etwas"; and meanwhile falls back upon the old notions of Charcot, Potain, and Fränkel, that the pain is due to the cardiac ischæmia; that it is the cry of the nerves in the heart for blood. Broadbent, like Dr. Mackenzie, saw that these explanations, spasmodic or quasi-spasmodic, must fail, and that "the ideas of those who have employed the term have been very vague." In his direct fashion Mackenzie concludes (*Diseases of Heart*, p. 304): "I admit I have no definite idea of the state of the heart during the paroxysms." Thus baffled as we are, is it then wise to resent the proposal on my part of a new hypothesis as an eccentricity—in the bantering words of a friend of mine, one far greater than myself, a "childish eccentricity"? Nay, surely, if there be a field open for a fresh eye and a new hypothesis, it is here.

Reduction of Contractility.—Dr. James Mackenzie [1] has offered us partial conjectures rather than a complete hypothesis on the causation of angina pectoris. He supposes that in angina the heart's nutrition by long strife has been much impaired, and its fibre degenerated; but he finds notwithstanding that in angina four of the properties of cardiac muscle may be, and usually are, intact, while that of contractility he assumes to be reduced; and for the determination of this single defect he relies upon the pulsus alternans. But, if the finger be an adequate witness, the pulsus alternans is not constantly nor even generally present in uncomplicated angina. However, Dr. Mackenzie denies the sufficiency of the finger for this observation. Still, how is this opinion reconcilable with what he admits—the mitigation of angina by enfeebled systole or mitral regurgitation, and its return on a *rise* of pressure? [2] And when he adds that exhaustion and shortness of breath on exertion are further evidences of this defective contractility, he is still travelling outside the argument;

[1] Mackenzie, Jas., *Brit. Med. Journ.*, Oct. 7, 1905.

[2] On the disappearance of angina with hyposystole see also Curschmann, in the discussion on this disease at the Kongr. innere Med. of 1891.

for neither of these cardiac symptoms is characteristic of angina. Nor do I know why Dr. Mackenzie assumes that in angina pectoris the heart has "been long exposed to excessive strain," and is more or less worn out; this assumption is not justified in pathology; in most cases of angina in middle or young life—as in the many syphilitic cases—the myocardium is quite healthy; and we have seen that even in elderly subjects the heart may still be good enough. Nor is it easy to accept his statement[1] that angina "is never the outcome of an acute affection; that it is invariably led up to by a long period of gradual exhaustion." Surely that it appears like a bolt from the blue in many a man apparently healthy and vigorous, and not only in acute cases such as the influenzal and the syphilitic than which none are more typical, is a matter of daily experience, an experience graphically described by Osler. That in many such cases stealthy degenerative changes have long been silently at work is true, but these are not necessarily of the kind of exhaustion; the myocardium may be far from exhaustion, or even from impairment. Pallor, faintness, and "collapse," with dilated pupils, may be frequent features of angina, as they are of biliary, renal, or intestinal pain; but the ordinary interpretation of these phenomena in angina is that the pallor is due to vasoconstriction, and that syncopic conditions act rather in the way of relief than of aggravation. Collapse, as Capps's and Lewis's experiments indicate (p. 478), is one of the exceptional effects of intrathoracic irritation. But to concentrate our attention upon defect of contractility; Dr. Mackenzie says, "with confidence," that angina "can occur when excitability, conductivity, rhythm, tonicity, are unimpaired," and when he proposes—as something must be wrong—to throw upon the opponent the proof that contractility is maintained, the reply is that, so far as we yet know, maintenance of contractility may be as compatible with angina pectoris as the maintenance of the other four qualities, or any of them; and until the high or relatively high arterial pressures, which frequently occur in angina, are found to be characteristic of impaired contractility, our anticipations will follow other lines. Take, for example, from the author's own

[1] Mackenzie, Jas., *Diseases of Heart*, p. 44.

careful record, the instance[1] in which during the attack the blood pressures rose from between 118 and 138 to 240-300; and as the pain subsided the pressures fell. "In another observation a rise to 160 presaged an attack." I agree that this rise was probably due to a large vasoconstriction, but what about the contractile power of a heart able thus to master, or cope with, increases of resistance so large. As Dr. Colbeck has remarked, the quality of tonicity, being a later development than that of contractility, probably would succumb before it.

One element of angina indeed there is, which may give rise to degrees of loss of contractility; namely, the cardio-inhibitory. Gaskell and His both described contractile reduction as a result of vagus stimulation; and it is possible that diminished conductivity may simulate, and practically amount to, defect of contractility. However, Rosenberg,[2] in summing up this part of the argument, confesses that "no hypothesis will serve which depends on reduced efficiency of the heart's muscle ('Verminderung der Leistung des Herzmuskels'); this condition in later stages may appear with dyspnea ('Lufthunger')."

When in his book on heart disease Mackenzie says (pp. 40 and 42) "the phenomena of angina pectoris are not the outcome of the gross lesions found, but are evidence of exhaustion of the heart's contractility"—though without any reduction of tone, he adds (p. 54), although "this exhaustion is the essential cause of angina . . . it is not dangerous," "and the phenomena disappear with the restoration of the nerve force." Then what is the danger element—the gross cardiac lesions? But often enough there are no such lesions, or not in sufficient degree of themselves to cause or menace death. And, as to the pain, we know of no severe pain from contractile exhaustion of this muscle. The arrest of the heart, in a week, in a year, or in a decade of years, may be due, as the attacks themselves may be due, to a "summation of stimuli"; indubitably the pains do depend upon an exalted state of the sensory paths; but the stop is not the angina, nor can it be an essential part of it, for plenty of people pass through the tribulation of angina and come safe out of it, not a few indeed safe and sound, while

[1] Mackenzie, Jas., *Heart*, vol. ii., 1911.
[2] *Loc. cit.* p. 310.

others after suffering for many years—twelve, fifteen, or twenty —die in the end of associated or intercurrent disease. Of the gravest maladies angina pectoris is one of the more curable.

Dr. Colbeck, in the article [1] to which I have referred, an article which contains an able criticism of the hypotheses of angina pectoris, illustrates many of these confusions of thought; he points out that not a few writers, by talking in the same breath of painful distension or dilatation of the heart and of cramp, blow hot and cold. His own opinion seems to be that angina pectoris depends primarily on coronary disease; but that the mechanism of it lies, not, as Mackenzie suggests, in a general defect of ventricular contractility, but in partial losses, in the inequality of contractile efforts in a muscular mass of which some parts are impaired; so that the unevenness of the pull hurts the weak places. This opinion approaches, or is identical with, that of some German authors. Von Romberg, after admitting that we cannot predicate dilatation of the whole chamber as the cause of the pain, mentions the conjecture that larger or smaller portions of the wall may be twitched thus unevenly. Yet, as in the partial cramp hypothesis, it is difficult to comprehend, whether on experimental or anatomical evidence, such differential rates of motion, or that the ventricle can act thus fractionally; or, in case of local buckling, that portions of the wall could be driven outwards time after time, without giving place to aneurysm or to some pouching visible at the necropsy. If, as Dr. Colbeck suggests, the degenerate patches are carried along in uniform motion with the rest, the discord may be painful, or may not. But after all, as Dr. Knott forcibly observes,[2] "these cardiac notions cannot account for the central forms of midsternal pain, which I have observed to be the most severe, as was the experience of Heberden himself"; though it is true no doubt that, cardiac and aortic innervation being similar in plan, in cardiac distress there may be, besides the "precordial" weary ache, also some ache in the shoulder and left arm, but, so far as experience yet tells us, with little intensity; rarely such as to cause much complaint. The truth is, quibble about it as we may, *cardiac disease is not characteristically painful.*

[1] Colbeck, *Lancet*, March 31, 1903.
[2] Knott, *Amer. Med.*, Oct. 27, 1904.

Claudication.—Now let us turn to that which is just now the most popular of the current explanations of angina pectoris; namely, to Potain's notion of "intermittent claudication" of the heart; a notion I have pondered with no little perplexity. I have read carefully what Potain, Pal, Sir William Osler, Dr. Parkes Weber, Obrastzow of Kiew, and other supporters of this hypothesis have said about it, also their tributes of priority to certain of our predecessors, such as Sir B. Brodie and Allan Burns; but so far I have failed to get to close quarters with their meaning, or to reckon the debt which we owe to any of them. On the phenomena of this claudication in horses, as described to us by Charcot, and on its interpreting value in explaining transitory aphasias, and other intermittent failures of function, in this area or in that, we have all been fully instructed; but, as I have said on sundry previous occasions, I fail to see their relevance, either by kinship or analogy, to angina pectoris. It is true that in the reports of senile cases angina pectoris often happens to be associated with diseased coronary arteries, but so are all other diseases in old people; even in angina the evidence of this proportion is founded upon the partial figures of the mortal cases. We have seen that no inconsiderable number of cases of angina—perhaps most of those ending in recovery—have been independent of coronary disease; and even in mortal cases I have quoted no indecisive post-mortem evidence to this effect (p. 374). Still, for the moment let us confine ourselves to the ordinary mortal case. Now these, we all agree, are generally attended with coronary disease, because the coronary disease is apt to determine, not indeed the angina, but sooner or later the lethal effect of one of its strokes. In these cases, as in the horse cases, we have, no doubt, a special incident of arterial disease, a sudden failure of a muscular organ supplied by diseased vessels. But with this coincidence surely the parallel ends. Arteriosclerosis of vessels distributed to muscular organs is common, yet intermittent claudication is something of a curiosity; however this may be one of those exceptions or eccentricities which now and then throw happy side-lights on more familiar processes. But does it? Does this claudication, as described in the horse, resemble angina in any but the most irrelevant manner? Surely the main feature of

intermittent claudication is claudication; the horse stumbles or falls, the limb of the man is for the moment maimed. How are such attacks to be compared with attacks of angina pectoris in which, unless it be the last, the heart, coronary anæmia or no, may be, often is, undisturbed; or shows at most no more perturbation than might be caused by a little flatulent dyspepsia, or by the irritation of a peripheral sensory nerve? Obrastzow says [1] that angina arises thus: the heart quickens in rate, the sclerosed arteries cannot expand quickly enough to meet it, ischæmia sets in with a shorter diastole, and so claudication; and this is angina! He calls it "dyspragia cordis dolorosa"! Every link in this argument is frail. The heart's pulsation need not, often does not, rise in rate; the coronary circulation is correlated not with the rate of the heart but with its metabolism and the blood pressure. But Dr. Parkes Weber, who pleads for "claudication," admits that the pulse in angina is often unaltered. The horse, as he trots along, suddenly drops, often with painful cramp of his legs; then after a few minutes, as muscular irrigation is slowly restored, he picks himself up and ambles forward, again to fall in the same way. The heart of an atheromatous and anginous patient trots along, and some day may stop suddenly; but it does not stumble, or not until in death it stumbles once for all. The heart cannot, like a pair of hind legs, curl up, wait for supplies, and then start away again. To suppose a spasm of these vessels is gratuitous. It is true we cannot say that the heart in angina never "claudicates," but when it does there is an end of the case: the halt is the halt of death. And yet Potain used to talk of angina pectoris as "painful *intermittent* claudication of the heart" (italics mine). But what of the fits of angina—the vast majority—in which there is not even a trip? Are we to call them painful arteriosclerotic cramp? But we have learned that in the heart muscle cramp cannot be, or rather that in its systole cramp is normal to it. And claudication is not due to vascular spasm but to obliterative arterial disease. As to the stumbling ("dyspragia angiosclerotica"), we have seen how the more observant clinical physicians have marvelled at the serenity with which the heart during the passage of the storm, over it

[1] Obrastzow, orig. art. in *Zentralbl. f. Herz- u. Gefasskrankheiten*, April 1912.

or through it, ambles evenly along. Some years ago I spoke of the heart as the one impassive agent in a torture chamber, and the phrase has been accepted. Almost in the words of my earlier papers, Gouget,[1] in a casual allusion to angina pectoris, has said, "It is obviously very illogical to attribute to intermittent claudication of the heart a syndrome during which this muscle proceeds in its function in an absolutely normal manner, without any modification of force, or of regularity . . . being present thus impassively at a painful and agonizing crisis." Where then is the "dyspragia"? And in these claudication cases we must not forget that there may be, and often is, an associated neuritis of the limb. One of Dr. Parkes Weber's patients, in whom this affection occurred in one leg only, had suffered in that leg from sciatica. The concurrences of cramp of the leg with angina pectoris in certain elderly atheromatous subjects, as recorded by Ortner and others, certainly prove nothing in respect of the nature of angina, and presumably are mentioned as analogies only. Many elderly persons are the subjects of cramp. If I seem to deal somewhat curtly with a hypothesis which has such distinguished adherents it is in no spirit of disrespect, but because by some mishap I have failed to comprehend it.

The *neurotic* conjectures of angina pectoris are ancient and manifold, but too vague and perplexed to detain us long. Laennec was a champion of some nervous hypothesis; he suspected the vagus. Trousseau compared angina with an epileptiform neuralgia. Stokes, and after him Bamberger, speculated on some cardiac hyperæsthesia with hyperkinesis. Lartigue[2] and Lancereaux sought it in the cardiac plexus; and Romberg, Williams, Friedreich, and others followed them. Bulb, phrenic nerve, vagi, intercostals, cardiac plexus, all have had their advocates and their day; to-day the vasomotory system is taking its turn. The clinching argument of these nervous advocates is that they are right because, such are the complexity, variety, and obscurity of the nervous endowments of the heart, they can neither be proved wrong nor be expected to explain how they are right. We will consider first A, the vasomotor hypotheses, and then B, the plexus hypotheses.

[1] In his treatise on "Arteriosclerosis," 1907.

[2] Lartigue, *Mém. sur angine de poitrine*, Paris, 1846.

A. *Vasomotor Hypotheses.*—On the vasomotor phenomena in these, as in many other sudden disturbances of functional equilibrium, I have spoken in other chapters. By perpetual vacillations of the balance of the circulation, they may play no inconsiderable, if an accessory, part in bringing about the discomfiture of a frail ventricle. However, they play a part in another hypothesis, that of Dr. Francis Hare.[1] After demonstrating the improbability of current conjectures, Dr. Hare argues that angina pectoris is due to a painful distension of the mediastinum, and this to intense vasomotor constriction in very large areas elsewhere. He thinks moreover that it may be by this congestion that the coronary arteries suffer injury, and the cardiac plexus irritation. Dr. Hare is coming nearer the truth; if he will substitute tension of the investments of a diseased aorta for tension of the mediastinum, I think he will find some things become clearer which he now finds obscure. But we are agreed that "high tension" is no necessary coefficient of angina. Ord and others have suggested that angina is due to a hypersensitive or labile state of the vasomotor centre — to which the cardiac centre is in close proximity—whereby the systemic arterioles are thrown too readily into spasm (a condition such as Salis-Cohen's vasomotor ataxia) by contingencies, mechanical, dyspeptic, or toxic (tobacco, etc.). Dr. William Russell, of Edinburgh, has substantially adopted this suggestion.[2] These storms need not be mortal unless the heart be somehow bad; and the hypothesis does not avoid the confusion between functional storm and grave organic disease (Chap. II.). Dr. Russell does not describe the effective mechanism, but the mechanical part of his explanation seems to fall into the category of the ventricular dilatation hypotheses. Sir W. Osler, who favours the notion of vascular spasm, regarding it as "the best explanation of anginal pain," wavers in a spirit of rather ironic meditation between various modes in which, with one's eyes shut, one may conceive its interferences; whether by a general constriction "increasing the tension of the pump walls," or by local (coronary) constrictions "disturbing the tension of

[1] Hare, F., *Med. Record of New York*, Oct. 28, 1906.
[2] Russell, W., *Brit. Med. Journ.*, Feb. 10, 1906.

sections of the heart," or by " pain producing resistance to tension by the muscle elements." In respect of a labile vasomotor centre taken alone, many of us, who have been only too familiar with the violent vasomotor oscillations of malaria, yet have not observed that these, or any consequent morbid susceptibility of the vasomotor centre, have ever issued in angina pectoris. That vasomotor constrictions, like muscular effort, chill, or any other influence on arterial pressure, are frequently the immediate determinants of the several anginal attacks, we are all agreed ; and it is probable that, as the medullary centres are more irritated, they become more labile, and attacks more frequent ; but in respect of the nature of angina, these storms have no more than an incidental importance. From the point of view of Nothnagel's assertions concerning these vasomotor factors, Dr. Mackenzie [1] has re-examined some of his own cases, taking a large number of sphygmographic tracings, and a few blood-pressure observations ; his results I may summarise in these few words, that in six cases of attacks in bed there was no sign of vasoconstriction in any one. He inferred vasoconstriction of any great extent to be an exceptional incident. In a case in which I was consulted by letter, an old gentleman, subject to angina but receiving much relief from pain by rest in bed, complained, while admitting this amelioration, of " heat rising from the chest upwards, which renders sleep impossible. . . . There is no rise of temperature." I guessed that irritation arising in the aorta, less than would generate pain, might, by setting up a vasomotor dilatation, arouse the brain which should have slept ; so that his convalescence was retarded.

Migraine is frequently brought into comparison with angina, and arguments used from a supposed affinity. But this is to argue *incertum per incertius* ; we know little of the pathogeny of migraine, we do not know even the mode of the pain, and its vasomotor conditions are very variable ; sometimes they are constrictive, sometimes expansive, sometimes neutral, but apparently always secondary. In Sir L. Brunton's own case I understand that the trunk of the temporal artery is distended, as it is, enormously, during the attacks in another friend of mine, perhaps as an effect of constriction in its distal

[1] Mackenzie, Jas., *Heart Disease*, p. 46.

ramifications. An area of constriction cannot be an area of high tension. In these parallels there is little to help us to explain the problem of angina.

Tides of vasoconstriction seem to be frequent in tabetic cases, not only in syphilitic angina and equivocal phases but also in the gastric crises, etc.[1] The link not infrequently noted between tabes and angina pectoris is of course syphilis (*i.e.* intercurrent syphilitic aortitis). Dr. Morison[2] thus sums up his own experience on this point: "Peripheral vascular spasm as a preliminary or primary event in the history of an attack in angina pectoris is, I believe, rare. As a secondary and aggravating event it may occur, as also may a condition of vascular laxity due to inhibited ventricular systole." These storms then have but an incidental importance in the problem of angina.

However, the neuralgic, neurotic and neuritic speculations on the origin of angina cannot be all set aside as visionary. To say, as one author says, that the pain of angina is "purely neural" is either meaningless or a truism. Assuredly we cannot feel pain without the assistance of nerves; yet, as Fagge argued, angina is too brief in its attacks, too immediately dependent on effort, and too mortal in its effects for a mere neuralgia. Neuritis of the vagus, no very rare event in syphilis, which has had some occasional advocates as a cause of angina, cannot of course hold the field long enough even to come into discussion; with the effects of vagus section or irritation the symptoms, save that of death, have little in common; and the notion or surmise that "the angina pectoris of diabetes" (whatever this may particularly be) is due to a neuritis of the vagus,[3] being no more than an unsupported and indeed improbable guess, cannot claim much attention.

B. *The Cardiac Plexus.*—Some thought must now be given to the hypothesis, not first suggested by Baumés (1808), or Gintrac in 1835, but by Wall,[4] championed later by Lartigue and Lancereaux, Peter, and Romberg,[5] and recently approved more or less by Groco and Fusari,[6] and by Dr. Mott,[7] that irritation of this

[1] *Vide* Heitz and Noréro, *Arch. des mal. du cœur*, sept. 1908.
[2] Morison, A., *Lancet, loc. cit.* p. 99.
[3] *Lancet*, July 1904.
[4] Wall. In the *Med. Trans.* vol. iii., 1772.
[5] *Leçons de clin. méd.*, Paris, 1880.
[6] Fusari, *Rev. de méd.*, July 1886.
[7] Mott, F., *Lancet*, Sept. 16, 1905.

plexus, by tension or by inflammation, might be the source of the pain of angina, and also of the reflex by which a healthy heart might be menaced, and a degenerate heart stopped altogether. On the evidence before us, all I have hitherto been in a position to urge is that the most frequent lesion of angina is a penetrating inflammation about the spring of the aorta. Desportes, Lancereaux, Peter and others, accepting a lesion so seated, supposed it to act by some implication and irritation of the cardiac plexus, an opinion at first sight attractive enough. Wall, quite in modern fashion, supposed a primary affection of the cardiac plexus, spreading thence to the corresponding spinal segments and beyond them; while Desportes supposed rather a neuralgia of the vagus. Lancereaux's first case was published in 1864;[1] to reinforce it he published a second.[2] The first case, in a soldier æt. 45, was apparently syphilitic; the second, in a man æt. 34, was attributed to malaria. In these cases the plexus was involved in active proliferating inflammation of the adventitia and inwards, and I shall show that this inflammation was indeed the cause of the angina. In his attribution of angina to disease of the cardiac plexus Lancereaux is quoted as if he had described an inflamed plexus only, but the careful reader will find more than this; he mentioned disease of the aorta on its external aspect, about the spot where the plexus lies against it. His cases were interesting; one, as I have suggested, was of syphilitic nature, for he describes the well-known prominent cushion ("plaque saillante") several centimetres wide, composed chiefly of connective tissue of new formation. The external coat of the aorta all around, but especially at the level of its adhesion to the pulmonary artery,[3] was the seat of "an extremely rich abnormal vascularisation," *and filaments of the cardiac plexus were embedded in a kind of plasma on the surface of the external coat* (italics mine). The coronary orifices were narrowed to the size of a fine probe, but the heart as a whole was "sain." He quotes, but without definite report of the state of the plexus, two other cases of

[1] The full title of his paper is "De l'altération de l'aorte et du plexus cardiaque dans l'angine de la poitrine" (*Gaz. méd.*, 1864; *vide* p. 432).

[2] Lancereaux, *Bull. de l'Acad. de Méd.*, Nov. 19, 1864.

[3] Here I may remind the reader of angina pectoris in mitral stenosis (*vide* p. 443), and in pericarditis of the membrane around the great vessels (p. 457).

necropsy of angina in which the same part of the aorta was found diseased. Vaquez, who is forced to admit with Josué and myself that angina may depend on lesion "at the origin of the aorta," explains it as stretching or inflaming the cardiac plexus or its branches. Barié rather jumps at the same explanation, or coquets with a "pseudo-angina"! (p. 953 of 3rd edition). Yet *he admits the symptoms of aortitis to be the same*, and that death may ensue! Other pathologists have declared the cardiac plexus in cases of angina to be normal. Dr. Morison [1] has published, with careful drawings, the necropsy of a case of angina of seven years' standing. The coronary arteries were calcified; in a twig of the right was "an intra-vascular aneurysm, internal to the muscular coat," and close upon it was a well-marked ganglion cluster. The valves were normal, and the myocardium well preserved. There is no note of the aorta. The discovery was remarkable, but we can scarcely suppose these ganglia to be sensory nerve organs. At present, although Krehl says the functions of the cardiac plexus and its associated cells, fibres and ganglia are unknown, yet most physiologists, with Langley and Anderson,[2] regard them as in function wholly motor; and they are probably right. Recently Dogiel [3] made a minute study of the intracardiac ganglia and of the several fibres of the vagus, and says the intracardiac ganglia consist of motor cells. This statement is so important that I give Dogiel's own words: "Meiner Meinung nach müssen alle Nervenzellengruppe gleichwoll sie im Herzen von Frosch, Hunde, oder Menschen liegen als motorische Nervenzentren des Herzen aufgefasst werden." He quotes Bidder and Lörit to the same effect: "Sämmtliche Ganglienzellen des Herzens physiologisch gleichartig sind, und stellen das motorische Zentrum des Herzens dar." Hence the motor capacity of every bit of the heart.[4] Between these and the vagus terminals there seems to be an "interference" as in the spheres of light or sound waves. If the vagus stimulus be prolonged the cardiac motor ganglia regain predominance, as they do if atropine be given. At any rate it appears that

[1] Morison, A., *Lancet*, Nov. 1902, and "Cardiac Pain," p. 23.
[2] See also Koch, *Med. Klinik*, 1912, No. 8.
[3] Dogiel, *Pflügers Arch. f. Physiol.* Bd. cxlii., 1911.
[4] See also papers by Kauffmann in *Pflügers Arch.* Bde. cxlvi. and cxlvii.

extension of disease to the plexus is unattended by pain.[1] F. Marchant and A. W. Meyer,[2] make the further important point that certain ganglion cells on the hinder and upper surface of the auricles between the openings of the venæ cavæ, form a mediate station of both vagi, whence they exercise inhibition on all parts of the heart. How far any such lesion of the cardiac plexus might thus be the cause of Dr. Mackenzie's defect of contractility in angina, if such there be, is a curious enquiry which we cannot confide to hands more skilful than his own; but it is *a priori* probable that with alterations or destructions, complete or partial, of the cardiac plexus, there should be associated some loss of one kind or another in the cardiac economy, though probably on the motor side only.

As to the notions of paroxysmal neuroses, neuralgias, hyperæsthesias, hyperkineses, and so forth, they seem to me to be patter, too unsubstantial either for adoption or refutation; they are of the stuff which the author of *Nightmare Abbey* called "philosophic gas." Parallels are drawn, as we know, between angina pectoris, epilepsy, migraine, and other paroxysmal pains; and if Sir James Goodhart (*loc. cit.*) is impelled towards some such unsubstantial view of angina it is because he is quite clear that the cause of it does not lie in the heart. But the coincidence of such affections with angina in the family or person, syphilitic convulsions being omitted, is, so far as my experience and reading go, no higher than the mean; and resemblances in the salient features are surely superficial. Angina is paroxysmal it is true, so is whooping-cough; but its intermittences probably depend more upon mechanical than on nervous variables, and to a great extent can by mechanical means be controlled. The vasomotor fringe in all of them may, probably would, present similar characters; but of such are not the deeper affinities on which we shall found classifications.

That a disease comparatively speaking so rare and so peculiar as angina pectoris should depend upon a perversion so ordinary, and in its nature so fluctuating, as a *retention of uric acid or other waste*, or of some chronically irritating *poison in the myocardium*, or, as others suppose, poisoning the cardiac plexus, is

[1] See Stienon of Brussels, *Arch. des mal. du cœur*, septembre 1910.
[2] Marchant and Meyer, *Pflügers Arch.* Bd. cxlv., 1912.

but guessing and seems improbable. "Ptomaines from the bowel" would probably reduce arterial pressures.

To Dr. Verdon's [1] argument that the origin of angina lies in the stomach, I have alluded (p. 344), and shall return to it in the section on treatment. That a windy stomach may aggravate and even determine a seizure, or death, is common experience, and therapeutically important; but this is not the root of the matter. Like the vasomotor phenomena, this is but an incident, an incident common to angina and many cardiac affections. And to compare the fixation of the chest and diaphragm in angina with that position in vomiting certainly does not go beyond analogy. He describes a case in which after a meal a full stomach would not cause an attack until the patient began to move about. Notwithstanding, Dr. Verdon has done good service by his emphasis on the gastric factor in angina; and, by his boldness in passing the stomach tube—a perilous procedure with an over-sensitive vagus?—has shown not only how gastric colic may start an attack but how by relief of the stomach it may be dispersed.

The Author's Interpretation.—*Angina and Aortitis.*—Hitherto, in the destructive part of this essay, I have found little difficulty. Some of the competing hypotheses were mutually destructive, others were inherently perishable. *Ipsae periere ruinae.* But now that I have to address myself to construction, I have myself to give hostages to fortune. In the midst of a tangle of prepossessions and incompatible or contradictory notions, let us try to see what nature is displaying to us, and displaying in vain. It is sure to be dark if we shut our eyes. From what has gone before we comprehend that angina is frequently associated with cardiac and other complications which confuse our vision of the disease in its essential form; but it is at least as true of angina as of other diseases, as indeed it is true for all scientific investigation, that if we desire to get a clear view of a problem we must, in the first place, select instances as free as possible from contingent interference. If we are to interpret angina pectoris we must begin our lesson, not with those records which are over-written, interpolated, or corrupted, but,

[1] Verdon, W., *Lancet*, June 18, 1910, and other papers.

as again in other scientific investigations, *per viam exclusionis*, with the simplest instances. Now has this rule been observed? Have not authors, on the contrary, sought their test cases, even by preference, from amongst those complicated with cardiac disease of one kind or another? One eminent physician, by way of a ready entrance upon his enquiry, at the outset put down "coronary atheroma, myocardial degeneration, aortic disease, aneurysm, high arterial pressure," and a few more dilapidations, as parts of his problem! Involved in such complexities, how can we see our way to separate the invariable from the incidental antecedents and associations of the process? To advance then *per viam exclusionis*, we shall, on the contrary, collect from examples of the disease, typical in their clinical aspect, those in which the pathological conditions, precedent and associated, were not the most but the least complex; so that, if possible, a common denominator may be found. Now the simplest forms of angina are those which arise—usually from infections—in persons young enough to be presumably free from primary cardiac defects. And of these instances there are a plenty, including data from necropsy, on which to found a solid hypothesis. I have cited many of them, and many more can be selected.

I repeat then that for proof, as for disproof, we must rely on cases selected for simplicity; cases stripped, so far as may be, of accretions. Even death is not an essential but an incidental issue of angina pectoris. If, in the many cases of relative simplicity whose nature has been happily veiled by convalescence from our scrutiny, we cannot give a demonstration of the pathological focus, yet in life or in death the clinical series is usually unmistakable enough for the discerning physician who will but use his eyes and forget his prepossessions. He will convince himself that a disease which not infrequently passes away, especially in young persons, cannot have its primary seat in an irreparable lesion of vital parts—such, for example, as obliterative disease of the coronary arteries or myocardial decay. Its causes must be compatible with at least relative recovery, and yet be such as may prove swiftly mortal. Now in an inflammatory or subinflammatory lesion occupying the suprasigmoid portion of the aorta, with its

investment, these conditions are fulfilled. Suprasigmoid syphilis is the most instructive process of the kind which may be accompanied by angina. In this infection we find an infiltration of the coats of the vessel, usually extending, in anginous cases, from the adventitia (see Aortitis, p. 183). On the inner surface we may see a cushion-like elevation of the wall, often of more or less annular disposition; this, if untreated, will soon extend downwards to the coronary mouths and to the valve, producing perilous or incurable mischief. The older the patient, or the more this kind of damage, especially in the coronary area, the more probable is sudden death; the less able is the heart to withstand a sharp inhibition.

Arterial Pain.—On cardiac pain, whether myocardial in seat or not, I have dwelt at length (p. 387), and have argued that in character it is not anginal. Dr. James Mackenzie, expressing the common opinion, says [1] "the heart muscle could give rise to pain just as much as any other muscle in the body"—and of such is angina. Well, instead of speculating on what could be, let us concentrate our observation upon what we know. Ridding our minds then of "could be," and fixing our eyes upon what is, we may perceive that there is one principal spot, lesion of which is ordinarily, if not exclusively, found in angina. The spot is the ascending aorta, especially the spring of the arch a little above the valve. This susceptible spot was first indicated by Morgagni who, on a typical case of angina, probably syphilitic, in a woman aged 42, says of the necropsy that the arch of the aorta was diseased and dilated, and acutely reflects "satis causarum in aortae vitiis habemus." The observation next in time of which I have a note is by Wall [2] who, in the necropsy of one of his cases, demonstrated a lesion at the origin of the aorta above the valve, and this lesion, taken it is true with disease of the aortic valve also present, he considered to be a sufficient cause of the angina. After him, as we shall see (p. 427), Corrigan made a similar observation, and arrived, independently, at the same conclusion. But, it may be urged, is the aorta, are arteries, capable of pain? The answer is certainly in the affirmative; and not in respect of an inflamed vessel only; though somewhat similar

[1] Mackenzie, Jas., *Lancet*, March 21, 1908, p. 856.
[2] Wall, *Med. Trans.* vol. iii. p. 12. Case XVI.

dense fibrous structures, such as a sinew, may in inflammation give rise to exquisite pain. The throbbing of a healthy artery, especially of the aorta, when so far relaxed as to expose its investment to the full force of the beat, becomes, as many a nervous orator knows, vexing or even painful. If it is true that arteries, like viscera or sinews, may, if not stretched, be pricked, cut, or tied without pain, yet in thrombosis, or at the moment of an embolism, an artery may be intensely painful, and during the consequent inflammation be exquisitely tender to pressure. A graphic story is told by Pierce of Bath of an arterial embolism in the Rev. R. P., who came to Bath in May 1684. While "very healthful, lusty, and strong, an intolerable pain seized him on a suddain in the calf of one of his legs, in so much that, hearing no gun go off, he had thought that somebody had shot him with a crossbow. . . . the acuteness of the pain made him sweat and faint, and very sick at stomach." I read lately of a case in which embolism of the femoral artery caused a screaming agony; and a few years ago I heard of a popliteal case in a cricketer, who turned angrily round to see who had struck him, and seeing no one very near him was amazed. The probability is that the pain in these cases is due to stretching of the outer investment of the vessel above the plug. Balfour speaks of "those atrocious pains which attend compression of an artery for aneurysm." In a case, reported by Dr. Bell, of sudden thrombosis of the abdominal aorta followed in a few days by paraplegia and death, the first symptom was a sudden and intense pain in the abdomen and back. The paralysis promptly followed. For a fuller consideration of such accidents I would refer to a discussion, "*Über Gefässschmerzen*," at the K. K. Gesellschaft d. Ärzte in Wien, 1893, published in the *Wiener klin. Wochenschr.*, Nos. 46 and 47 of the same year; and to cases vividly narrated in Osler's *Lumleian Lectures*. The aortic lesion, if angina is to spring from it, must in my opinion penetrate to a certain depth, or begin on the outward side, and be, one might suppose, at least of subacute activity, though, as with fibrous adhesions elsewhere—as about a joint—the part thus stiffened or adherent may, after the subsidence of inflammation, remain sharply sensitive to tension. That aortitis, without any cardiac or cardiovascular lesion, can

and does set up anginous pain is now denied by none but those hardy advocates who, against the evidence of fully typical clinical symptoms, not only withhold this name, but, if it be adverse to their own postulates, scout the evidence of these parts laid bare at a necropsy. Jean Heitz admits that chronic aortitis may produce severe retrosternal pain, and also the peculiar dread, yet not only declines to call this angina, but warns the reader against the use in "coronary angina" of baths which he does recommend for the aortitis. As he prudently avoids differential criterions he puts the doctor in an awkward dilemma. Bäumler lets himself go so far as to say (incidentally in a recent paper) that angina pectoris may arise either from disease of the coronary arteries or of the root of the aorta ("Aortenwurzel"); but a well-known scientific canon constrains us to think not how little a particular cause can be made to explain but how much, and to mistrust a multiplicity of causes.

Still, it is demurred, aortitis, or at any rate atheroma, does not always produce angina, and when it does the reason is that in the suprasigmoid area the lesion is very apt to encroach upon the coronary arteries; whence the angina. With the part of these vessels in the matter I have dealt sufficiently (pp. 353-381). As regards the aorta, this first portion seems to be the most sensitive (p. 416), and the lesion may break into angina when it penetrates to a certain depth; down, as I suggest, to the outer fibrous investment of the vessel. If Thoma be right in his belief that he had found Pacinian corpuscles in the walls of the arteries (p. 418), the mechanics of pain in these structures become clearer to us. We have remarked that in any part of the body tension of fibrous structures, such as the periosteal or fascial, is a potent cause of pain; a more efficient cause than the tension of muscular structures. In skeletal muscle cramp gives pain of this kind because it tells upon the fibrous elements and tendons with their well-known sensitive end-organs; but of cramp, as we have seen, the heart is incapable. So in angina, tension is probably the mechanism; and from this point of view we may regard the ascending aorta as the tendon of the heart. An inflammatory infiltration may hurt after the manner of a small suppuration within or beneath a fibrous band, and a succession of deposits or "poussées"

may bring about several series of attacks; in acute aortitis this may be the mode of the seizures. But in the long reiterated attacks of chronic cases we may have to do rather with the element of sprain, or morbid tension; I compare it again with the drag of adhesions so familiar to us as a cause of pain on the movement of a joint once inflamed and subsequently tied by adhesions. Yet what is an old ankle sprain to a sprain of an aorta, which is not only an arch upon which life is supported, but also a bearing which is never out of action, a limb which can never know a moment's rest upon the sofa! But here, in order to carry the reader with me to this new point of view, I must be permitted to make a digression.

This digression is *to examine briefly what is known of the physiology of the cardio-arterial afferents*; unfortunately, in contrast with our knowledge of surface afferents, our knowledge of deep afferents is slight. In this difficult enquiry I have been happy enough to obtain the aid of Dr. Hugh Anderson. After the reading of a paper by myself at the Cambridge Physiological Club on February 10, 1909, it appeared that, by the clinical path, I had been approaching certain views which Dr. Anderson had already attained from the approaches of physiology. What these are I will try with his assistance to indicate; but it must be understood that he is not responsible for my imperfect description.

Afferent machinery is that which carries impressions from the surface to an equilibrating centre; it is an informing in mediate contact with a distributing system. An afferent unit then is an outward cell connected by a conductor with such a centre; and an abstract diagram of such a system may be expressed as a sphere coated with sensitive cells attached to a corresponding number of radial fibres converging to a central office. But this diagram expresses perception only, an apparatus which effects nothing. For action, that is for meeting the readjustments, limbs are necessary, and the uniform sphere has no limbs. For external limbs the sphere must be reformed; extended here and there, and shaped to various instruments: and on the whole the mechanical instruments will be outside, those of the generation of energy within, though not altogether; for instance, the valves of the heart are mechanical limbs yet are

within. To make such limbs then, and to arrange that the large and various parts concerned with adaptations shall lie towards the world, while those concerned with the generation of energy shall be stowed within, the outward coat of our sphere must be drawn out, and tucked in, on as many different ways. As the animal series develops, continually new and new extrusions and intrusions must be made for this packing of certain parts inside, and for room and play to the parts which are to be without.

Now with the outward limbs and their efferent conducting threads we have not to do; we are at present concerned only with those packed inwards, and of these especially with the heart and great vessels; parts in their origin diverticula from the digestive tube, and therefore to be packed inside. Speaking generally, with every extension of outward limbs — such as arms and legs—there must have been some corresponding inward movement of the vessels, as of the other organs of vegetative life. A conspicuous example of this dragging of parts away and together is seen in the tangle of fibres known to us as the brachial plexus, a perplexity caused by the dragging outwards of the upper limb and of these, and other, strands with them. Thus, to return to our abstract sphere, for every extension of the surface outwards some corresponding area of it dives inwards.

Now we remember that a large parcel of our sphere consisted of sensory units, each unit being a cell of the surface attached below to a conducting centripetal fibre; when therefore a sector of the sphere is drawn inwards we shall see with the eye of the mind that each unit thus affected must be dragged, cell and fibre, inwards; and, as the packing in and growing out become more and more elaborate, we shall find our afferent units penetrating in and in, carried farther and farther into the interstices, more and more intricated with other shifting and crowding parts and strands, until in their disguise we utterly lose sight of their migrations and origin.

If then we are to recognise those afferent units which have thus been shifted and hidden within, we must try to track them in these modifications; either by dissection or by embryological methods, or by both together. We must look for a sometime surface cell, derivatively a cell of the cutaneous system, attached to a conducting fibre. Now Dr. Anderson has pointed out that

such units, masked as they may be by change of place and fashion, are to be discovered. The "touch corpuscles" described by Pacini, and by Vater, or indeed by Malpighi a hundred years earlier, are endowments of the skin itself as we know it—the integument still external. But their successors perceived that like bodies are to be found far below the skin, namely in the tendons and many other places; and that each of these sensory end-organs is a part of a compound unit of the kind we have been contemplating; namely, a nest of cells each with its centripetal twig. Moreover, embedded in the midst of the body, the same apparatus may be detected even in its unit form, namely, as a single cell and twig; and between this unitary form and the enclosed complex of Vater, many intermediate and not a few transitional forms may be observed. Now, from what has gone before, we shall anticipate that these apparatus also, whether in unit or compound form, will always be associated with the prolongations of connective fibre to be regarded, both ideally and practically, as processes of the skin sweeping and twisting, however far inwards, with the inpacking of the viscera to which they are attached. In this sense then all sensation, inside and outside, is skin sensation, and is still of the same nature and mechanism. Conversely, no part which is not originally of the skin can feel.

Once more; by the work of Head, Mackenzie and others, we know how manifold is cutaneous sensibility, as in the kinds of touch, in temperature, superficiality, depth, and so on; differentiations which have been developed with the different experiences of the skin in its many wanderings and exposures. Leaving on one side epicritical and other qualities belonging to surface as surface, if we follow the processes of the skin into the tucks which we have been pursuing inwardly, we shall find that they have dropped, or never become possessed of, the sensory qualities correlated with surface experience; their attributes are more rudimentary, and as a matter of fact may be confined to sense of tone and pain, the one being normal, the other abnormal. Tone and pain in this use are probably but degrees of the same quality; pull, for instance, being the means of those marvellous harmonies of muscular use so fascinatingly illustrated for us by Professor Sherrington; pain being illustrated by the same function in excess, as in muscular cramp.

When within our bodies all organs are moving in harmony, and so all the pulls balanced, we feel well; when their functions are not all in equilibrium the tensions here or there are unequal, and we feel these discords of pull or squeeze, from slight uneasiness to intolerable agony. Dr. Hertz, in his *Goulstonian Lectures* of 1911, made this true and pregnant remark, "I believe that tension is the only cause of true visceral pain." And, conversely, wheresoever these pulls and squeezes generate vibrations such that they are carried to the sensorium, there, even in remote recesses, the afferent quasi-cutaneous end-organs and their conductors, mediate or immediate, will be found.

Now is this sketch, curt as it had to be, sufficient to place us at the right point of view to understand how in fact the heart and great vessels may behave themselves in respect of sensation and pain? We have seen that our knowledge of the deep afferents of these organs is defective, and our opinions are rather anticipations than knowledge. But with some certainty we may say that the investments of the heart and arteries are involutions of the skin, are indeed subcutaneous structures; in them therefore, so long as and wheresoever the need for afferent currents exists, we may assume that the machinery has not fallen into an atrophy, and will moreover be of the same general form as in the skin of other parts; that we shall find in or about these investments the cells and afferent fibres, whether single or compound, to which we have alluded as end-organs, or under the name of Vater or Pacini bodies. That the need for such afferent impressions does still exist in these organs, in the heart and arteries for instance in order that the fluctuations of tension within them may be regulated, is beyond doubt. The pulmonary circulation is not subject to very large fluctuations, and on this side of the heart we should not expect that the afferent mechanism would be very evident; it is on the systemic side that we should expect them to be chiefly manifest and, on this side, particularly at the spots where tension is most liable to become strain; namely at the transference of tension into velocity at the issue from the heart into the aorta. Here degrees of resistance to the initial velocity would be of the greatest importance to the well-being of the circulation, and here therefore regulation most necessary. Here, that is

at the entrance of the aorta, we should expect to find the most sensitive area, and the most efficient afferent mechanism. And, as these bodies are of cutaneous origin and seat, we should first seek them in the investment of the ascending arch of the aorta. As Dr. Anderson has pointed out, it is at the neck of the heart and aorta that the indrawn skin is, as it were, "pinched into folds." But the search for bodies of this kind in the elaborated frame of man, or even of the child, when they have been overgrown by the close interweaving of other structures, is very difficult; and we can scarcely be surprised that hitherto they have eluded much of the search which may have been made for them. The search must be made in the fœtus, and along the lines of animal evolution.

Bruns and Genner (Marburg)[1] have studied this sensitive area of the aorta—the pressure tambour as I have called it, the relations of the depressor to the heart's work, and the aorta's elasticity. To every rise of pressure in the aorta they found the depressor sensitive. If in animals the depressors were cut out, there was no compensation, or but very transient, so that in its absence the heart was readily strained.[2] Now Professor Danilewsky of Cracow,[3] in the course of certain researches upon revival in the mammalian heart, found an application of the electrodes to the root of the aorta to be followed by arrest of the beats; therefore this part has been called the "heart-knot." If, during an electro-cardial tracing, this part be pierced, the auricular contraction may cease; then, as the animal dies, the ventricular contractions wane and the intervals between them increase. I need not insist upon the importance of these facts in respect of my hypothesis of angina pectoris. Again, Keith and Ivy Mackenzie[4] allude to the "abundance of the nerve supply to the sinuses of Valsalva at the commencement of the aorta and round the auriculo-ventricular orifices, which they regard as end-stations for sensory

[1] Bruns u. Genner, *Deutsche med. Wochenschr.*, Nov. 17, 1910.

[2] One curious result of cutting the depressor I may record, though in the text irrelevant; namely, that Gigou and Ludwig, and again Bruns, found that after a while the heart hypertrophies. See for both articles, *Arch. f. exp. Path. u. Pharm.* vol. lxix., 1912. Bittor had said ("Blutdr. u. Herzhypert. bei Aortenskl.," *Münch. med. Wochenschr.*, 1907) that in man suspension of depressor action was hard to verify, because the heart worked up to it.

[3] Danilewsky, *Arch. für Physiologie*, 1905, Suppl. Bd. p. 190.

[4] Keith and Ivy Mackenzie, *Lancet*, Jan. 7, 8, 1910.

reflexes." It is not unlikely that what I may call the anginiferous area, though here most sensitive, is not strictly limited to this part, but may extend along the arch beyond the origin of the great vessels, and even to the abdominal aorta and cœliac axis. To apply the electrodes to the root of the pulmonary artery produced, in Danilewsky's experiment, not an inhibitory but an augmentor effect. Thus if it be true that angina may originate in the pulmonary artery (p. 301), we may suppose that no case on this circuit would, as angina, prove mortal.

This much then we can say, with some certainty, and on other evidence contained in more than one part of this chapter, that, whatever the mechanism at the neck of the heart and aorta, here is a knot of exalted sensibility to tension. It is my position that angina pectoris consists in a morbid exaltation of this sensibility to tension, felt in the majority of cases at this spot, or more or less onwards in the vessel, rather than in the heart itself; and that according to homology the mechanism for this irritation should be seated not so much in the inner coat of the aorta as in its quasi-cutaneous investment. By this mechanism every wave of tension should be propagated and distributed according to its indications, so that pressures in excess and in defect may be equalised or compensated. It would appear that, however large, these fluctuations—and in vigorous bodily exercises we know that they may be very large—are normally thus redistributed without troubling the sensorium; the balance is automatic. It is when by disease the receptive surface is made abnormally sensitive that these stimuli force their way, as pain, to the sensorium. Happily the sensorium does not usually become well aware of excessive tensions, say of Bright's disease or of aortic regurgitation, so long as the investment of the vessel remains normal; but if the investment of the ascending aorta—and perhaps of more extensive areas of the compound cardio-arterial vessel—be by some morbid cause thrown into a hypersensitive state, then we receive, as it were, heated reports of blood-pressures, and tension darts into pain, pain which in the coverings of other viscera, in pleura, meninges, peritoneum and so forth, we know only too well may be grievous, but which, when arising in the citadel of life, is terrific. And, for multiplication of the pain,

when the corresponding sensory centres, and centres again above and beyond these, by accumulation of stresses have reached a certain level of tension, any small additional stimulus suffices to overthrow equilibrium. Hence the importance in the treatment of angina of a period of complete rest long enough to calm this central lability. When by the assault the patient is dismayed, the efferent nerves of the heart, receiving this false impression, may, as in labyrinthine disturbances, act with a readjustment as false, but from the nature of the case with far more serious consequences. If the action of the heart be unduly augmented no great harm may ensue, good perchance; what does matter is the awakening of the inhibitory efferent, which, as we shall see in another section, by an over-protective action only too often, in saving the part, sacrifices the whole.

Unfortunately when from these general considerations we pass on, still relying upon Dr. Anderson, to apply our anatomical knowledge of these nerves, we realise how defective this is. We have reason to believe that afferent fibres pass from the heart by way of the vagus, and that the depressor branch plays a specially important part in the co-ordination of the intracardiac and systemic blood pressures. We know that the cardiac branches of the stellate ganglion contain afferent fibres, and we may reasonably suppose that most or all of them are passing from the heart to the spinal cord by way of the upper thoracic nerves. Further, Luschka and other anatomists have shown that the phrenic nerve is connected with the lower ganglia of the cervical sympathetic and also gives off branches to the great vessels and to the pericardium.[1]

But, as I have said, about the distribution and mode of ending of these nerves there is much doubt. Smirnow,[2] who has investigated the sensory endings in the heart by the methylene blue method, says that many sensory fibres end in the endothelium; but that many others end freely as brushes of fibres in the connective tissues. He has also examined the nerves of the heart after previous section of the vagus, or depressor, and has been led by such observations to the conclusion that some of the free

[1] See also Leonard Kidd's researches on the phrenic nerve (*loc. cit.*).

[2] Smirnow, *Anat. Anzeig.* Bd. x.

endings in the heart are connected with depressor fibres. On the other hand, Köster and Tschermak,[1] using Marchi's method for tracing the fibres degenerated after section of the depressor nerve, say that this nerve is distributed to the aorta and does not enter the heart. Neither Smirnow nor Köster and Tschermak describe end-organs in the heart itself, and we do not know in what way the phrenic branch to the great vessels ends; but we have noted that Thoma[2] described Pacinian corpuscles upon the thoracic aorta and other vessels; and Rauber[3] found these bodies in man upon a branch of the phrenic nerve between the pleura and pericardium on the left side (see p. 454 on pericardial angina). Dogiel reports that his pupil Schemetkin discovered "sensibler Plättchen" in the adventitia and other parts of the aorta; and Stadler reports these bodies in both the thoracic and abdominal aorta. More recently Manouélian,[4] who accepts my hypothesis, has demonstrated, in dogs, sensory end-organs on nerve-fibres amid the elastic muscular and connective tissues of the aorta, whereby

[1] G. Köster and A. Tschermak, *Archiv f. Anat. u. Phys.*, 1902 (Anat. Supp.).

[2] Thoma, *Virchows Archiv*, Bd. xciii. p. 100, and Bd. xcv. p. 335, writes: "Another very interesting element in the structural elements of the adventitia are the Pacini or Vater corpuscles. Their very frequent occurrence in the parts about the aorta (as also in the mesentery and elsewhere) is well known. I have been able to discover them not only in adults but also in children, and in the fœtus in large numbers, *in the outer layers of the adventitia of this portion of the aorta and in the connective tissue layers immediately contiguous.* They are seated for instance on the anterior surface of the aorta, and very thickly in the neighbourhood of the origin of the cœliac axis and of the mesenteric artery, as also in other vascular areas." When this article was written Thoma had not traced them in the thoracic aorta; but in the following year (*vide ibid.* Bd. xcv., 1884, p. 335 note) he reported that, besides the parts of the arteries mentioned in the previous article, he had discovered the Pacinian bodies "in the thoracic aorta also" (and in the subclavian and some other arteries).

[3] Rauber, *Untersuchungen ü. d. Vorkommen und Bedeutung der Vater'schen Körper*, München, 1867, p. 4. "As with the skin over the surface so a papillary sense-membrane ("eine papillöse Gefühlsmembran") extends through the depths of the body, with manifold interruptions and recommencements, with many offsets. This membrane must be recognised as an organ of sense." Rauber then remarks upon the identity of the end-organs in two regions of the body so different; and points out that, while those on the skin are sensitive to many outer influences, those within are adapted especially to pressure perception. The abundance of sensory end-organs in the mesentery is familiarly known.

[4] Manouélian, *Acad. des Sci.*, 16 juin 1913; quoted *Zentralbl. f. Herz- u. Gef.-Krankheiten*, Sept. 15, 1913; and *Ann. Past.* xxviii., juin 1914. See also Hüber, "Sensory Endings in Viscera," *Journ. Comp. Neurol.*, 1900.

he explains the contractions and dilatations of the vessel. And I see that Bechterew (p. 247) assumes their presence.

It may be well however, before discussing the pains associated with disease of the heart, to consider what the meaning of the " free-endings " in the heart may be.

Dr. Franklin Evans, in his minute description of nerve end-bulbs in the joints,[1] says that these are often intimately connected with the small arteries of the part—in the adventitia ; and occasional encapsulated bulbs of arborescent endings are applied to one side of the vessel, parallel to the stream.

In the more primitive forms of animals all the afferent nerves are connected with sensory epithelium on the surface, after the diagrammatic type already imagined ; but in higher forms some of the epithelial cells become detached from the surface epithelium and, retaining their connection with afferent fibres, form free sensory end-organs in the connective tissue. Simple forms of such end-organs are represented in the bird by the Grandry and Herbst corpuscles, in both of which the termination of the sensory fibres in connection with cells may be clearly demonstrated. The Pacinian corpuscle of the mammal is also represented in very young animals by a group of cells among which an afferent nerve-fibre branches. But in the adult the cellular origin of a Pacinian body can no longer be detected and the nerve-fibre appears to branch " freely " in the core of the corpuscle. Further, the sensory nerves of the muscle-spindle or tendon-organ, as shown by Howell and others, appear in the adult to end freely in knobs upon the muscle-fibres or tendon-bundles ; but a study of these organs during early development again points to a termination of the afferent nerves in cells upon the muscle-fibres or upon the tendon-organs. It seems not improbable, therefore, that the so-called " free-endings " found in the connective tissue of the heart may originally have developed in connection with epithelial cells which have afterwards been so far altered as to become indistinguishable ; and it is interesting to notice that Smirnow mentions special endings in the chordæ tendineæ resembling those found in the tendons of the amphibia.

If the views here put forward (Dr. Anderson's) be correct, we must regard not only the Pacinian corpuscles on the vessels but

[1] Evans, F., Thesis for M.D., Cambridge University, May 29, 1912.

also the free-endings in the heart as sense-organs of epithelial origin. But, whether this view of the origin of the free-endings be accepted or not, it is important to recall to mind that we have no histological evidence of any direct connection of sensory nerves with the *muscle* of the heart or of the vessels. *I would urge then that any pain caused by undue distension or contraction of the heart must be assumed to arise from the tension exerted on the connective tissue investing these organs, and to be conveyed from the connective tissue by the nerves that end "freely," or in end-organs, within it.* Similarly we are told, on high surgical and physiological authority, that pain in the renal area arises from two parts only—the pelvis and the true capsule.

Moreover, the connective tissue investments of an organ, such as the heart, which has been displaced during the course of development from the neck to the thoracic cavity, are derived from more than one region, and their sense-organs, as well as the sense-organs in the connective tissue of the thoracic cavity, would be dragged down in the connective tissue with the heart. Accordingly, we are not surprised to find that the phrenic nerve, which supplies the skin in the neck, sends branches also to the pleura and investments of the great vessels, as well as to the diaphragm, a muscle derived from the cervical region.

But though such sense-organs may be displaced far from the skin from which they were originally derived, the central end of the afferent fibre connected with each sense-organ will always retain its central connections with associate cells in the spinal cord, and these connections it will have in common with the afferent fibres coming from the parent area of skin. Hence irritation of an outlying sense-organ, say upon the phrenic nerve, would excite the conducting group of cells common to it and to the skin over the shoulder, an area which is supplied also by sense-nerves arising from the same spinal roots as the phrenic nerve.

On this account, if this common path be kept in a state of excitement by irritation of an end-organ, say on the phrenic nerve,[1] we should expect to find that slight stimulation of the area

[1] I am again indebted to the paper on "The Phrenic Nerve" by Leonard Kidd, *Rev. Neurol.*, 1911.

of skin over the shoulder would give rise to an undue disturbance in the conducting cells, and to transmission of exaggerated and possibly painful impulses; and, possibly, even without any additional impulses started from the skin itself, there may be a feeling of pain over the shoulder because the whole of the conducting cells associated with that skin area had been so excited by the impulses incessantly reaching them from an outlying sense-organ originally derived from the skin area, that painful impulses were started and transmitted to the brain from the common conducting group, and in consequence were referred by the brain to an access of painful impulses from the skin itself. Tidy [1] says that in aneurysm just above the Valsalval sinus, right shoulder pain may occur; and Kidd has seen the same pain in cases of aneurysm. If the aneurysm be of the transverse arch, left shoulder pain may occur.

This is, of course, what is meant by the term referred pain, and by the association between segmental pain in the skin and internal organs, so ably investigated by Ross, Mackenzie, Head and others. In the often quoted paper of Ramström, although it is true that in his summary, and therefore in all reports of the paper, the author does not mention, and apparently does not quite realise that, in all his experiments on manipulations of the intestine, when afferent reactions were awakened he was dragging on the mesentery; whether by traction with a blunt hook, by raising the bowels upon a pad, or otherwise. When drag was carefully avoided, mechanical or hot or cold touch called forth no response. It is agreed (Johnston,[2] Ramström, Lennander, Mackenzie [3]) that the serous surfaces are themselves insensitive, the end-organs *being distributed in their investments* (areolar substructures), endowments in which the mesentery is peculiarly rich. Ramström himself says that when he pulled upon the mesentery *the pain would get worse for* 1-2 *minutes after the cessation of the pull, and might not subside for several minutes.* This accords with our clinical experience of angina. Gray and Parsons [4] speak in the same way of the pain and in-

[1] Quoted by Leonard Kidd (*loc. cit.*).
[2] Johnston, *Brit. Med. Journ.*, Sept. 11, 1907.
[3] Mackenzie, *Brain*, vol. xvi.
[4] Gray and Parsons, Arris and Gale Lect., *Brit. Med. Journ.*, May 11, 1912.

hibitory effects of a drag on the mesentery, and affirm that to handle the gut itself with a careful avoidance of any drag on the mesentery, produces hardly any vasomotor disturbance. This important contrast they repeat three or four times. They add that traction on the stomach and œsophagus excites an almost pure vagus inhibition on heart and respiration, and a fall of pressure; with very little vasomotor effect. *Strong traction may stop the heart.* In operations on the stomach and under the diaphragm the splanchnic system is hardly disturbed. Vagus inhibition predominates, and this in accordance with the effects of flatulency in cardiac disorder (see Capps and Waters, p. 478). As Professor Keith says, "*mesenteries and visceral ligaments come into action only when the limits of normal movement are reached.*"

After this digression let us return to *angina pectoris and aortitis.* In the severer cases of infectious aortitis the adventitia presents to the eye inflammatory swelling; it is thick and swollen with exudate, cell proliferation, and vascular congestion (*vide* p. 158); while the grey translucent elevations on the inner aspect turn later into syphilitic or atheromatous spots and patches. No cause of angina comes more clearly into view than syphilis, and to Wilks and Welch we owe the description of syphilis of the aorta (and other arteries and viscera) which has been elaborated, with scant acknowledgment of their forerunners, by Heller and the Kiel school (Doehle, Saathoff, etc.) (p. 170). Now syphilis attacks the adventitia, and thence penetrates inwards as, or associated with, a "meso-aortitis productiva." On this process, and the clinical features of this disease so seated, I hope the able description of Dr. Mitchell Bruce may receive the attention it merits.[1]

Aortitis of this kind or degree may kill by cardiac inhibition, especially when it checks an old or diseased heart; occasionally, even when the heart and the coronary arteries are intact (*e.g.* p. 376, Case XII.), it cuts the thread. Now, after many years of direct observation and of special search in the records, with the exception of certain rare cardiac cases to be discussed presently, *I have failed to discover a case of unmistakable angina in which, whatever the disease of the coronary arteries, the aorta, on careful examination, was demonstrated histologically to be inwardly*

[1] Bruce, Mitchell, *Lancet*, July 8, 1911, p. 72.

and outwardly sound. The notes of the very few cases alleged in books and essays to present such conditions are all lacking at this crucial point; the pathologist, being by preconception blind to its essential importance, omits or slurs over in the aorta the investigation he makes minutely of other parts. In a recent public discussion Sir William Osler [1] quoted a case of angina in which "nothing abnormal was found." This case I shall consider presently (p. 439). Briefly, the aorta (its inner surface only was mentioned, and of this only its naked-eye appearance) was said to be smooth and free from all disease; but I have heard since that the appearances were not quite negative; that the exact words are "except some slight atheromatous changes in the aorta." The coronaries at any rate were normal. Now let me turn to a parallel case, which points a moral. At a discussion on this subject at the Royal Society of Medicine [2] Dr. Archibald Garrod, in combating my aortitic opinions, quoted the case of a man, aged 40, who died suddenly in a second attack of angina. After death pneumococcal endocarditis was found, with recent plugging of the mouth of the right coronary artery. So far as he could remember there was no disease of the aorta; therefore the angina was due to the coronary plug. But Dr. Garrod is always careful to verify his facts; and before the *Proceedings* were published, in the month following, he wrote to me to correct his report, saying that "the arch of the aorta was somewhat dilated and the thoracic aorta very atheromatous"; a condition, at aet. 40, probably due either to the pneumococcus or to syphilis, both of them among the causes of infectious aortitis. Now, that coronary embolism may produce angina pectoris I shall not deny, these cases are reserved for the moment; all I wish by this citation to do here is to point the moral of careful observation and careful record. The suprasigmoid part of the thoracic aorta, which, I repeat, is a sort of *sensitive tambour* to regulate cardiac energy by the mediation of the vagus, is the most prone to produce angina; the lesion must be acute, subacute, or issue in tensile adhesion; and it must penetrate deeper than

[1] Osler and Bullard, *Medical News*, Dec. 15-22, 1909. I have been unable to obtain this paper.

[2] *Proceedings*, March 13, 1908.

ordinary atheroma usually does. To be content with a glance at the lining only of the vessel, to belittle any patches and streaks on its surface, sometimes perhaps to overlook even these, and always to overlook its outer coat, is perfunctory work. If our fathers examined bodies with less instruction, it was with less prejudice. In T. Paytherus's case (a port wine bibber aet. 66) Parry reports not only the state of the heart, but adds that two inches above the aortic valve the vessel was atheromatous and doubled in thickness. From without then the vessel should be examined even more assiduously than from within, for certainly a periaortitis may give rise to angina; here signs of acute inflammation may be found, or in a chronic case the adventitia, especially in old syphilitic cases, may be altered: on section the vessel, probably dilated a little, if no more, will feel dense and gristly to the knife, and may be adherent. Unquestionably angina occurs in many a man whose slowly degrading vascular system one would not readily accuse of activity in any area; yet, as we shall see presently, I, and others, have found that even in these slow forms of atheroma subacute phases of local extension are not uncommon, or adhesions may drag; moreover angina, though not unknown in the aged and decayed, is prevalent rather in senescence.

Of syphilitic aortitis Stadler (*loc. cit.*) says that one case in five had suffered from angina, minor or major; Josué has accepted my views in their entirety; and indeed our German colleagues are now beginning to talk of *schmerzhafte Aortensklerose*. Now if I consult the first handbook of medicine at my elbow, I find among the symptoms of aortitis these: "It may consist in a weight or oppression, or a severer distress, amounting to intense retrosternal pain, comparable with crushing by a red-hot iron bar; or a vice-like constriction, often accompanied by much tenderness or superficial hyperæsthesia. There is often also *a peculiar sense of dread, as if the last moment were come* (italics mine). From thence atrocious pains may radiate to the left arm or to the arms."

This aortitic agony Gibson contrasted with that of angina pectoris as a continuous and not a paroxysmal pain, an opinion in which I cannot follow him, unless the "status anginosus" of very intense cases be described as "continuous." He added quite

truly: "There is no considerable dyspnea, but death is often sudden. Or again, the oppression may never proceed to these extremities, but *continue rather as an anxiety without positive pain*, or may even simulate a hysterical state of mind" (italics mine). Boinet writes[1]: "In aortitis the pain is retrosternal . . . is sometimes compared to a bar compressing the chest . . . sometimes it is agonizing, extremely violent, piercing, atrocious, with accesses of painful dartings to the left shoulder . . . often in the left arm, down to the little finger. In their suddenness, their intensity, and their gravity, these attacks resemble angina pectoris. . . . They are sometimes followed by syncope, sudden death. An attack of angina pectoris may be the first manifestation of acute aortitis, especially above the aortic valve . . . when it does not depend upon obliteration of the coronary arteries . . . which event is observed more particularly in chronic aortitis." The attacks resemble angina pectoris! May we not rather ask the learned author in what respect these attacks differ from angina pectoris? Brouardel himself, who had studied aortitis, acute and chronic, chiefly from a pathogenic point of view, said of its symptoms—that they vary with the symptoms of the disease and other conditions, but that the most frequent of them is angina pectoris. And to his opinion let me add the comment on angina of that no less observant clinician, Broadbent, who wrote on disease of the aorta as follows[2]: "Angina, answering to this criterion, is met with clinically in association with aortic valvular disease, or with aortitis, or with dilatation of the aorta general or aneurysmal, or with atheroma of the aorta without obvious dilatation, or with fatty degeneration of the heart . . . but all these, *except perhaps acute aortitis* (italics mine), are very common without angina. Angina is established when the coronary arteries are implicated." Of course this last reflection had to be brought in as a tribute to orthodox opinion; but seeing that the author had just admitted acute aortitis to be the one state in which angina occurs unconditionally, and as this disease, however disposed to implicate the coronary orifices, does not always attain them,[3] and if it does, and even blocks their mouths,

[1] Boinet, in Brouardel's *Traité de médecine*; also *Angina Pectoris*, 1897.

[2] Broadbent, W. H., *Lancet*, May 27, 1905.

[3] See, *e.g.*, Coloured Plate by Dr. Mott in Allbutt and Rolleston's *System of Medicine*, 2nd ed. vol. vi. p. 568.

may not empty them of blood, and as coronaries are continually blocked or choked without angina, the inference of Broadbent's last clause seems not inevitable. The changes in the aorta giving rise to angina—the author goes on to say, as Gibson said, very truly—are often of a subacute degree, so obscure and insidious as not to reveal themselves by evident symptoms. Now these descriptions in the current text-books are found not, as the unwary reader may suppose, under the head of angina, or of coronary atheroma, but of aortitis; as any one who will turn to such works for himself may read.

It has often been objected to me that before we attribute angina pectoris to changes in the aorta, we must explain how a disease, comparatively speaking rare, can depend upon a morbid process so common that few senile aortas are innocent of it. To retort that the same objection arises in the attribution of angina pectoris to disease of the coronary arteries, which is almost as common, does not carry us much farther. We have to try to discover in the one case or in the other the factor which determines the particular issue of angina. I have suggested that this elusive factor may be a more active phase of aortitis, more especially of aortitis in a certain area (supra-sigmoid), or of a certain depth of penetration; and that atheroma is prone to such phases of recrudescence. Bureau [1] describes such phases thus: "Putting aside," he says, "cases of primary acute aortitis, in the immense majority of cases aortitis develops itself in aortas previously the seat of the chronic affection—as in gout, senile degeneration, and the like." Gibson, in his work on the *Heart*, expressed the same opinion, almost in the same words. Broadbent again says (*loc. cit.*), "the changes in the aorta usually begin so insidiously and chronically, that no evidence of them can be detected till dilatation of the vessel is discovered; but occasionally, if the aortitis becomes acute, it reveals itself by symptoms." Sansom [2] wrote, "'Only atheroma,' do you say? Examine it, and you will see some old and some new patches, in different stages, like an eruption above the aortic valve . . . some old and yellow, or calcified, or fibrous, but beside them little gelatinous patches,"

[1] Bureau, "Aortites aigues," Thèse de Paris, 1893.
[2] *Clin. Journ.*, Oct. 24, 1900.

and so on. Indeed these exacerbations are now generally accepted by pathologists; it is agreed that in chronic atheroma there are subacute periods when gelatinoid patches supervene, either in contiguous spots, or with a marginal distribution; patches themselves to pass likewise into "ordinary atheroma." [1] Now these more active atheromatous patches, under such conditions, resemble the primary acute patches of the acute infections; and if seated in a particular area, and penetrating to a certain depth, they will be revealed by similar symptoms—symptoms of the type which has received the clinical name of angina pectoris. Marmorstein, in an article on "Des aortites grippales," [2] gives as a chief symptom of the aortitis, "douleur avec caractère angoissante et rétrosternale." From his article I quote a case in point:

Male, æt. 50. Angina pectoris, with pulse of high pressure. Aorta dilated to 3 cm. in the right second space, systolic murmur, hammering second sound, apex thrusting. Died in attack. Necropsy: dilated ascending aorta. Internal aspect swollen, and covered with patches—some bony, others moderately hard and yellow, *others soft, grey, or reddish grey.* Left ventricle enlarged. Valves fairly normal and competent, but aortic cusps opaque. Diagnosis: chronic aortitis with acute *poussées*; one acute gelatinous patch close to orifice of left coronary, and probably the cause of death." (No allusion to outer aorta, no sections made of this part.)

I may mention here that in the case of Dr. Forrest's (p. 514) I find a note that the aortic arch, palpable in the jugular fossa, was very tender to the touch. Her attacks were severe and the seat of the pain mid-sternal, often darting into both arms. This I have noticed more than once.

In the month of May 1908, six months after the delivery of the lecture on Angina pectoris [3] which is the substance of this essay, while reading in Sir W. Osler's library I lighted upon a certain article by Corrigan, extracted from No. 35 of the Dublin

[1] The connection of gelatiniform patches with atheroma and with syphilis respectively is a problem for further investigation; but it seems clear that such patches are not peculiar to syphilis.

[2] Marmorstein, *Rev. de méd.*, 10 mars 1908.

[3] To a large medical audience at the Mount Vernon Rooms, Fitzroy Square, London, under the presidency of Sir Hermann Weber.

Journal of Medical Science. This distinguished physician commenced his article with these words: "I have two objects in view in these observations: first, to show that *inflammation of the lining membrane of the mouth of the aorta* (author's italics) is capable of producing the group of symptoms to which we give the name of Angina pectoris, and is therefore entitled to a place in the list of the causes of this formidable affection; secondly, to trace the pathology and treatment of aortitis." The paper is illustrated by a coloured plate of a heart and aorta laid open, "showing the diseased portion round the mouth of the aorta; the lining membrane being crimson red, turgid, and swollen out by an effusion of lymph behind it, to the height of at least one-eighth of an inch." (The author, unfortunately, omits to indicate to which case of angina the plate belongs; but if, as would appear, it belongs to Case I., this was of a carpenter, æt. 39, and is obviously and very characteristically syphilitic.) In Case II. a "gelatinous pulpy effusion under the lining membrane" occupied a space above the valve for a breadth of one-half to one inch. The valve also was invaded, and "one cusp ruptured, evidently recently." This case also looks like one of syphilis, but the age of the patient is not given. The heart in both these cases was "enlarged," but no histological examination of it is reported; nor is the state of the coronary arteries or their orifices alluded to. Both cases were marked during life by angina of the ordinary description, and severe in degree; and the aortas seem to have resembled closely that shown by Dr. Parkes Weber at my Fitzroy Square Lecture (p. 187), and afterwards at the Royal Academy of Medicine.

Corrigan adds a chronic case in which after death extensive disease of the aorta was found. For three years this woman, æt. 47, had complained of "*what was supposed to be nervous spasms*" (italics mine) (attacks of pain stopping her while walking, etc., etc.). On this the author writes, as again and again I have written (p. 241): "It is hard to resist the conclusion that had the fits, erroneously supposed to be nervous angina, been referred to their true cause, the inflammatory action in the mouth of the aorta might have been arrested, and life have been saved." The author's treatment was mainly a course of mercury, pushed till the gums were sore, and repeated later. The success

of this prescription in more than one instance—a common one in those days for any kind of "inflammation"—supports the presumption that some of his cases were syphilis.

Other cases he mentions, some with necropsies, but, either in detail of diagnosis or of pathology, less decisive; I need not follow the author through arguments with some of which I cannot agree. My purpose is to call to witness the only modern physician who—so far as I know—had distinctly attributed angina pectoris to disease of the aorta; yet even Corrigan does not attach angina definitely to disease of the aorta as the pathological counterpart of the clinical series, but says only that aortitis in the area in question is "capable" of setting up these symptoms, and is "entitled to a place in the list" of its causes. Once more I repeat the canon of scientific reasoning, that causes are not to be multiplied *praeter necessitatem*.

Broadbent, in another essay,[1] describing, as we shall see presently, a case of "aneurysm at the very root of the aorta, within the pericardial sac," comments on it thus: "that it is not always possible to distinguish between anginal pain due to aneurysm in this situation, and true angina." Then why be so bent upon evading the obvious inference? Broadbent's endeavours to find a criterion for the distinction, such as the course of its radiations, the degree of dread, and so forth, were in vain; these differences we shall all agree are no criterions. Gibson was very bold; he settled the matter by seizing upon the symptoms of aortitis and handing them all over to the heart. Of aortitis, he says, "the pain is frequently of the true cardiac (!) type; that is to say, it has the sternal position (!) and the tendency to radiate" along the well-known lines.[2] Another physician no less eminent is even more ingenious: he says "the anginous crises produced by the Coronaritis blend ('se confondent') intimately with the *painful incidents* ('*les accidents douloureux*,' author's italics) of aortitis": really a delightful apology!

The great weight of the evidence connecting angina with the aortic area is far from being as widely recognised as it ought to be. Austin Flint urged, as Norman Moore has done,

[1] Broadbent, W. H., *St. Mary's Hospital Gazette*, vol. i. No. 1, 1895.
[2] Gibson, G. A., *Diseases of the Heart*, p. 826.

that "in angina pectoris, disease of the aortic orifice and of the aorta is found more frequently than any other changes." The following case, taken from one of Heller's papers,[1] is very instructive. A man, in the course of a violent effort, broke down one aortic cusp, and *also forced out a pouch above it in the suprasigmoid portion of the aorta* (italics mine). The first, the most violent, and the abiding symptoms were attacks of angina pectoris, the pain radiating as usual down the left arm. These attacks beset the patient till his death six months afterwards. Dr. Norman Moore[2] has said, "When angina pectoris is present, even if on a particular examination a murmur be not discovered, disease of the aortic valve (*it would be safer to say, of the aorta*) (italics mine) is almost always present." Dr. Havilland Hall, who is not without sympathy for my hypothesis, has spoken to the same effect as Moore, whose clear-sighted maxim is well illustrated by the following case (from Vaquez and Bordet):

A man of 48 returned home one morning after a rackety night, and on lying down was seized by an atrocious attack of angina pectoris. Face pale, and anxious; respiration accelerated but superficial. No dyspnea. The attack lasted many hours, and an acute aortitis was diagnosed. Blood pressures were normal. Auscultation and percussion gave no assistance, but an orthodiagram, taken a few days later, revealed that the aorta was uniformly enlarged to a considerable extent; and its walls were thickened. The arch was not raised (see p. 204). (Probably a syphilitic case.—C. A.)

Again, in his *Lumleian Lectures* of 1909, Dr. Norman Moore said, "In cases of sudden death with disease of the aortic valves, the myocardium is sometimes degenerate, but in other cases shows muscular tissue in which degeneration has not begun"; and proceeded to contrast clearly with angina the pain sometimes "felt towards the apex" in mitral disease, rightly explaining that, even if severe, it is more a continuous ache (*vide* pp. 387 and 441). Nothnagel, confused as were his ideas of angina, nevertheless correctly made this same discrimination. Recent researches (Carey Coombs and others) show that rheumatic inflammation begins, not in the valve, but in the deeper structures around it. We

[1] Heller, *Deutsche Arch. f. klin. Med.*, 1903.

[2] Moore, N., "Hunterian Lecture," Oct. 10, 1900; reported *Lancet*, Oct. 20, 1900.

know that the a-v bridge may thus be injured (Mackenzie and others).

Angina Pectoris and Aneurysm.—Let us tarry a while longer to weigh some further evidence from diseases of this class. From what has gone before, it may be asked: Now, if angina pectoris depends upon a flaw in the thoracic aorta, is it found that aneurysm of this part is attended, in its initiation at any rate when it is forcing the vessel, by anginal symptoms? The answer is that in no inconsiderable number of such cases this is so; Dr. Gibson says, "in a large proportion of cases the initiatory stage is attended with such symptoms." Sir William Osler[1] says "severe attacks may occur in the early period of aortic aneurysm" (a period, by the way, when the coronary orifices are less likely to be occluded); "they were noted in the histories of 22 out of 132 cases." That such should always be the case is not to be expected; the site of the aneurysm, the rate of its growth, and other variables, determine the contingency. And weight or oppression at the chest, though consistent with angina pectoris, may be no warranty of it. The initiation of a thoracic aneurysm is often latent; or if at this period pains there be they may be vague and wandering, or fixed and tedious, rather than anginous in character. Boring and continuous pains are more indicative of a later stage, a stage of pressure on neighbouring parts, and of some considerable extension of an aneurysm which in its initiation may have been silent. Thus Gairdner, in his essays on *Aneurysm*, in my System (first edition) and in "Reynolds'," distinguished between the pains of pressure—pains gnawing and persistent, and "paroxysmal" pains. He quoted cases of referred pains of this kind, relating them to the districts of Head and Mackenzie, in paragraphs which I cannot quote at length; but, for instance, after many years of study and enquiry he was prepared to state that "in not a few instances in which the diagnosis from other facts is well established, aneurysm of the arch of the aorta leads to symptoms closely resembling typical angina pectoris . . . with pain and numbness of the left arm . . . extending sharply from the chest into the neck, face, shoulders, and down one or both arms." The angina, he adds, "may precede the discovery of aneurysm by years," and "usually

[1] Osler, W., *Medical Chronicle*, May 1906.

vanishes as it is established" (*i.e.* when the investment of the vessel has gone under). In one of Osler's cases (*loc. cit.*) the angina began in 1893, but in 1894 there were no physical signs: six years later the angina had disappeared, but definite signs of submanubrial aneurysm had come forward. Dr. Drummond [1] has since insisted upon the same features; and Dr. Graham Steell, in accordance with my own interpretation, states that angina is by no means rare in aneurysms involving the *ascending* aorta.[2]

About five years ago, with Dr. Glasier of Mildenhall, I saw a man, æt. 26, with angina. Patient graphically described the "grip" about the junction of manubrium and mid-sternum, and the radiation across the upper pectoral area to the left arm as far as the elbow. Syphilis was strongly denied, but we suspected aneurysm. The signs were a trifle of dulness to the right of manubrium, a systolic thrill strongest at the base, and a simultaneous murmur following the first sound after a brief interval. Heart distinctly hypertrophied. Arteries too palpable for age. In spite of the hypertrophy the blood pressures always ranged low—about 100-110. We provisionally diagnosed aortitis and aortic stenosis; possibly a Valsalval aneurysm. Treatment, chiefly by rest and iodide of potassium, had the effect only of mitigating the angina. Six months later Dr. Glasier reported to me that "he died in the night, bringing up a large quantity of blood. I think there is little doubt that he died from a ruptured aneurysm." Dr. Hawkins also saw this patient.

Two other recent cases in my own practice of syphilitic aortitis, running side by side, have gone on to aortic regurgitation; in one of them aneurysm has formed without angina; in the other no aneurysm is discoverable, but the angina is very definite, indeed rather prolonged and severe. Quincke [3] says that, besides pressure pains, thoracic aneurysm "may assume all the well-marked phenomena of angina pectoris." Trousseau, in his chapter on angina pectoris, which he was unhappily encouraged by Duchenne and Aran to regard as a neuralgia analogous to trigeminal neuralgia—his reason being that in some cases he had failed to discover any disease of the heart—described, notwithstanding, two severe cases, cases which he himself had distin-

[1] Drummond, D., *Brit. Med. Journ.*, Jan. 13, 1908.
[2] Steell, Graham, *Lancet*, Dec. 9, 1911.
[3] Quincke, Ziemssen's *Cycl.*, Art. "Aneurysm."

guished as genuine angina, which afterwards proved to have been initial stages of thoracic aneurysm.

A case of another kind I may quote from Dr. H. L. Dixon's thesis for the Cambridge M.D.[1] The case occurred at St. Thomas' Hospital, under the care of Dr. Acland:

Male, æt. 52. Arteries thick and tortuous; large white kidney and anasarca. High arterial pressure. Suddenly was seized by a typical attack of angina pectoris—his first. This was not repeated, but it was followed by severe continuous pain in the epigastrium, which lasted till his death three days later. P.M. Hæmopericardium. ℥ xii. One inch above the aortic valve was a transverse tear of the inner and middle coats of the vessel. The blood had dissected its way into the pericardial sac.

This case may be compared with the well-known record of Morgagni on the wife of a painter in Padua, æt. 40, who suffered from "violent pains in the chest and arms." Vallisneri invited Morgagni to open the body . . . "a little above the semilunar valve there was a narrow ulcer." Lancisi, in an unmistakable case of angina ("quemdam sub sterno dolorem usque ad humerum propagatum, et pectoris angustiam"), reports at the necropsy an aneurysm of the aorta, "sub egressu e pericardio." Desportes made a like observation, wondering why the disease in this place should set up angina; the pain he compared with that of popliteal aneurysm.

In another essay Gairdner remarks that the small aneurysm seated near the aortic valve is the kind more frequently associated with angina; his words are, "Small aneurysms thus impinging upon the heart are especially productive of angina." He says also, "Even small aneurysms, if arising very near the heart, and especially such as project into the pericardium or compress in any degree the base of the heart, are much more apt to give rise to angina-like symptoms"; and that in the presence of anginal symptoms the attention of physicians should be very minutely directed to irregularities of the ascending aorta. To draw again upon Sir William Osler's stores,[2] we read, "There may be paroxysms of pain, often simulating angina (why "simulating"?)

[1] Dixon, H. L., Act kept., Oct. 8, 1909.

[2] Osler, W., Clinical Lecture (in 1901) at Johns Hopkins Hospital on "Aneurysm of the Descending Aorta." For Rupture, see p. 207.

in small aneurysms at the root of the aorta; in fact there is a special group of cases in which angina pectoris is the initial symptom of aneurysm of the aorta." In Dr. Mackenzie's work on *The Pulse* (p. 239), the following case of angina is cited:

Male, æt. 40. Tightness across the chest, and pain down the inside of both arms, especially on exertion. Death sudden. P.M. a small aneurysm of the aorta immediately above the aortic valve. *Both coronary arteries were healthy;* but there was some "fatty degeneration" of the myocardium.

Once more, Breitmann,[1] in a careful study of syphilis of the arteries, says, "If, however, the aneurysm is situated near the aortic valve, the signs may be those of the valve lesion *with the anginal syndrome*" (italics mine). Now it is remarkable that in his descriptions of myocardial syphilis with dilatation, and of syphilis and thrombosis of the coronary arteries, Breitmann does not allude to this "syndrome."

A distinguished physician has warned us lest if angina occur with such an aneurysm we fall into the error of giving it its own name! Surely the error would be less in the bestowal of the name than in the ignorance, too often inevitable, of the full conditions of the diagnosis. It has been objected that in aneurysmal angina patients do not die suddenly as in the senile cases; but sometimes they do: however, as a rule, aneurysm occurs in comparatively young men with sound hearts.

Armed with quotations from authors so eminent and considerable, I need not multiply witnesses to the same effect. If here and elsewhere I am tedious, it is because only by reiteration can I press my argument home; with Lisette, "Je dis la même chose parce que c'est toujours la même chose." It is conceded that aneurysm of the thoracic aorta, and especially of the first part of it, without any cardiac or coronary disease, may set up angina pectoris; the pain being due to the aortitis or strain which precedes or accompanies it. If this be so, then the aorta alone is capable of producing this malady, and in a form, to use Gibson's words, "most typical, so that not the slightest suspicion may be entertained of the nature of the case; and which means early structural change in the wall of the aorta."

[1] Breitmann, *Gaz. des hôp.*, 21 fév., 1903.

If then to this rule of association of angina with aortic lesion we find some exceptions, and some I have yet to consider, we shall presume the variation to depend on conditions for the precision of which we have not as yet sufficient data; and until the attention of pathologists shall be bent to this problem, we shall not obtain the data we need for the complete morbid anatomy of angina. I repeat that no notes of such cases are acceptable in which the state of the first few inches of the aorta in its whole thickness is not accurately described as to its appearance, the exact site of any lesion, and its histology. Such careful reports as this of Dr. Walter Broadbent,[1] from which I will quote a few lines, are rarely to be met with (to Dr. Poynton's I have referred already, p. 275). The patient [2] was attacked by acute aortitis and severe angina pectoris, which ended fatally. Dr. F. G. Bushnell cut sections of the aorta and heart's muscle. "In the aorta there was a perivascular infiltration with leucocytes round the vasa vasorum of the aortic wall, more marked in the outer coat." In this case, as contrasted with those of Poynton, *the sections of the heart muscle* (*duly stained*) *showed a singular absence of change, whether in striation or nuclear or other alteration* (my italics). It will be remembered that in many or most of these cases we have to deal with degenerate, or with syphilitic hearts; and as in syphilis this area of the aorta is very prone to suffer, and thus to be the seat of angina, the cluster of symptoms will vary. In one case of the kind indeed, besides the sinus aneurysm, "atheroma of the aorta and coronary arteries" is mentioned; but aneurysms of the sinuses of Valsalva, till revealed by their comparatively early rupture and fatal issue, are often latent. Even Sir William Osler,[3] whose researches on this subject are of such importance as to make my incessant liberties with his name inevitable, in emphasising, as he does, the closure of the orifices of the coronary arteries as the chief factor of angina, gives, in my opinion, insufficient attention to the immediately contiguous part of the aorta. Corrigan, in one of his cases,[4] reports that "an effusion of organised lymph was found between the lining membrane of the aorta and the fibrous coat."

[1] Broadbent, Walter, *Angina Pectoris*, 1897.
[2] *Dublin Journal*, vol. xii., Nov. 1837.
[3] Osler, W., *Lancet*, May 27, 1905.
[4] The case is given more fully in the chapter on Aortitis, p. 427.

Precisely, in the very spot I have anticipated; I ask for no better evidence. My experience then coincides with that of Dr. Norman Moore and others, not forgetting Nothnagel himself, who says that, in contrast with mitral lesions, which are not attended with anginous pain, "*lesions of the aortic ring, on the other hand, are often painful, and attacks of true angina are common, particularly when the root of the aorta is involved*" (italics mine). I could not myself have written a passage more convincing of the general truth of the position I have taken up in the interpretation of this disease.

We know now that aortic valvulitis may penetrate below and beyond the valve, and may creep so far as to impair the neighbouring structures. Dr. Mackenzie, in conversation, described to me a case of acute rheumatism, with aortic valvulitis and angina, in which at the necropsy aortitis was anticipated, but none, as I understood, was found. Probably the inner surface only was examined, no sections made, and the investment of the vessel neglected; I suggest that the valvulitis had penetrated into the subjoining parts, perhaps to the pericardial investment of the aorta. Perisigmoid as well as suprasigmoid lesion may suffice to produce angina; this I shall consider later.

We have it on the testimony of Broadbent, and other trusty observers then and since, that although of course any one or more of its characters may be absent from a particular case of "true angina," yet in each and all of them the group is characteristic of both angina and aortitis. Dr. Lees, at the Harveian Society's discussion, agreed that a dilated aorta was a very important confirmatory sign of angina. I reiterate then, is it not strange, on the general acceptance of aortitis as a cause at least of "anginiform symptoms," that in necropsies on this disease a careful description of the suprasigmoid aorta should have been so frequently omitted—a neglect which in the records of many treatises on the disease I have had again and again to lament? Concerning the coronary arteries, no feature, however minute, is forgotten; the state of the heart's muscle is most carefully reported; but the aorta is either overlooked altogether, or we are put off with some such hasty phrase as "aorta atheromatous." Some of the older authors give us better data; for example, of one case of typical angina pectoris, Jurine reports

that the heart was negative; that the posterior coronary trunk was indeed calcified in its middle length but its orifice open to a probe ("stilet ordinaire"), and the anterior artery presented only an atheromatous patch = 4 mm. "But," he proceeds, "the base of the aortic cusps was of a cartilaginous consistence." In one of Jenner's cases the coronary arteries were diseased, but so also were the coats of the aorta; for two inches above the sigmoid valves they were doubled in thickness and studded with white patches. Exemplary instances of a more scientific examination of the parts are of two cases I have already quoted, one by Dr. Poynton, who leans to my view of angina,[1] and one by Dr. Walter Broadbent; in these cases *sections of the aorta* were made. In No. 1, F. æt. 38 (the "true case of angina" of Cheadle) (*vide* p. 275), the sections of the ascending arch showed extensive patches of acute inflammation. The intima was raised and pinkish, some translucent patches and a few small areas of atheroma were appearing; microscopically the usual infiltrating proliferative characters were seen, especially around the vasa vasorum; fibrous tissues swollen. In his second case the same minute examination of the aorta was made, and a similar state was found. I have alluded to the morbid sensitiveness of adhesions, well exemplified in inflammatory affections about the joints, but about the aorta I find but one description of this condition, nay, but one reference to it, this was by Hirtz and Braun.[2] In three cases of "classical" angina (syphilitic aorta) these pathologists described *dense adhesions about the aorta, binding it to neighbouring organs.*

M. Huchard's collection of necropsies, laborious as it is, fails of its full value, by no fault of his, from this imperfection of the pathological records, especially in respect of the suprasigmoid aorta. Although Huchard was possessed by the coronary idea, we do owe him the service of laying the ghost of "pseudo-angina"; as this bubble went on bubbling it was well that an enterprising controversialist was ready thus to prick it again.

Even in records of cardiac syphilis precise note of the suprasigmoid aorta is often lacking; yet, as we have seen, in these cases angina pectoris is far from uncommon, and is

[1] Poynton, *Heart Disease*, 1907, p. 241.
[2] Hirtz and Braun, *Soc. méd. des hôp.*, 31 mars 1911.

fairly amenable to specific remedies. In these cases of angina, if mortal, and a necropsy obtained, as in Dr. Parkes Weber's case [1] (*vide* p. 264), suprasigmoid aortitis is found, with or without coronary occlusion, but generally with it; as this is a chief factor, not of angina, but of death in angina. Indeed angina pectoris in middle or early middle life is so frequently syphilitic that this probability should be uppermost in the mind of the physician, so that by prompt and vigorous specific treatment not only may the angina be cured, but also destruction of the aortic valve prevented. Some of these cases of aortitis, assuredly syphilitic, progress destructively in spite of specific treatment, but this is nearly always established too late. A certain case reported by Broadbent (*loc. cit.*), which ended more fortunately, was probably of this nature:

Male, æt. 50, angina pains, never very severe. Next a systolic aortic murmur appeared, and the pulse, sometimes small and weak, sometimes large and lax, fell in number—to 60 and 54 (pressure upon or invasion of the auriculo-ventricular bundle?—C. A.); then came a soft diastolic murmur, not always audible, which at length disappeared. In six months all symptoms subsided, leaving the systolic murmur and a parchment-like second (aortic) sound.

Dr. Bruce Porter, of Windsor,[2] has described a characteristic case of the kind which he rightly diagnosed as "an acute aortitis" issuing in injury to the aortic valve. The patient was extremely ill, suffering from "anginal attacks." Maximum systolic pressure about 200. Kept going by morphia and atropin and nitroglycerine, which had to be increased to $\frac{1}{200}$ every hour, and the morphia to $1\frac{1}{2}$ gr. in 24 hours, for "only thus was the pain in the aorta kept at bay." The physical signs were those of "general dilatation of the aorta," then of aortic regurgitation, and ultimately of mitral insufficiency. . . . The patient at date of report was recovering, having had only three anginal attacks in the last five months. (No history of syphilis was to be obtained, but it is difficult to attribute it to any other cause.)

I deferred the discussion of the only case on record—so far as I know—of angina apparently genuine, and mortal moreover, in which after death no lesion was found, aortic or

[1] Reported to the Royal Acad. Med. in March 1908.
[2] Porter, Bruce, *Brit. Med. Journ.*, Nov. 21, 1903.

other (Osler and Bullard) (*vide* p. 423); this case, as reported, stands against every hypothesis of the disease. I have tried to get a copy of the original paper,[1] but none is to be had. I take the account, therefore, from Osler's *Lumleian Lectures*.[2] M., æt. 26: "a heavy smoker," and athletic. The pain is described as "in the heart"; which is not typical, at any rate in terms. He had an enormous number of attacks during the year and a half before his death. It is said that after death nothing was discovered; that every part of the heart and its appurtenances were normal, or practically normal. Now as yet this case stands alone, it stands without corroboration. Some fallacy in diagnosis or autopsy there may have been, for the record is contrary not only to every one of the multifarious conceptions of angina, but likewise to all clinical experience of the disease. No young man dies of nothing at all; in every death there is lesion or poison. That the man was a heavy smoker was noted as important; any one who has seen a bad case of tobacco angina can guess that it might prove mortal; though I have never seen nor read of such a case, with adequate pathological record. Against the supposition of syphilis an autopsy even apparently negative seems decisive. As I have said before (p. 425), the quotation may be inaccurate. However, until this case receives some corroboration it must stand as an enigma, and quotable only under reserve. When we look at the evidence as a whole, are we not compelled to say with Josué[3]: "All these facts are in disagreement with the coronarian hypothesis; on the other hand, they are readily explained by that of myself (Josué) and Clifford Allbutt. The pain of angina pectoris arises at the origin of the aorta, and is provoked by certain changes in this part of the vessel. Our case (a case he had been describing) favours this way of looking at the matter"?

Is there an Angina of the Heart?—My argument then so far is that angina pectoris is due to tension of quasi-cutaneous investments, not to anæmia of muscular tissues or strife within them; and that in the vast majority of cases

[1] *Medical News*, Philadelphia, vol. lxxvii. p. 974.

[2] Osler, W., *Lancet*, March 28, 1910, p. 840.

[3] Josué et Paillard, *Bull. et Mém. de la Soc. Méd. des Hôp. de Paris* (Séance, janv. 29, 1909). For his own previously published views Josué refers to the *Soc. Méd. des Hôp.*, 3 juillet 1908; a paper he kindly sent to me.

of ordinary angina this tension is exerted upon the thoracic aorta, whose ascending portion appears to be more exquisitely sensitive to tension than the rest. Furthermore I have been led to suspect that such tension and capacity for pain may be felt in the same or a similar manner, and propagate itself in the same or a similar manner, in this fibrous investment as it extends forwards upon the aorta or backwards upon the body of the heart itself. The innervation of this cardio-aortic area is so nearly uniform that pain of like origin and character might arise from capsular tension in more than one parcel of it.

Let us see then *if there be a pain, or pains*, local or referred, anginiform or otherwise, *which do signify distress at the heart itself*; pains which we may construe as a remonstrance by an overworked, engorged, inflamed, underfed, or decaying organ? On this point I think our daily experience leaves little doubt. We have all recognised a pain, notably in mitral disease, which cannot well be regarded as other than cardiac in origin; and in mitral disease it may be, I suppose it is, called forth by distension, or possibly by fatigue, of the heart. Sir William Osler says[1]: "In most affections of the heart pain is conspicuous by its absence. . . . Of valvular lesions mitral disease is often associated with slight pain, particularly in children with greatly enlarged heart. And sometimes in women *the pain is of great severity and persistence, but it rarely has the characters of true angina*" (italics mine). Pawinski again writes that the pain of "heart strain" is never anginiform, nor even violent, but a weary ache; and he thinks that if in any case, as in one of Da Costa's, the pain was more like that of angina, the case was not a simple one, but complicated with heart affections, perhaps with a pericarditis sicca. This vulgar heart pain then does not resemble that of angina; it is not vehement nor paroxysmal; it is never attended by the peculiar dread; and radiations, usually absent, are never remarkable. It is a dull, depressing, continuous ache. Whatever its source, this pain is an intercostal neuralgia, most intense—as is often the case—in the ramus perforans lateralis. Differentially speaking, Dr. Mackenzie[2] writes that the aching and hyperæsthesia (tender-

[1] Osler, Sir W., *Luml. Lect.* p. 841. [2] Mackenzie, Jas., *Heart*, vol. ii. 3, 1911.

ness of skin and muscle) in ordinary cardiac dilatation are around the apical area, and that as the distension is relieved they pass off. This pain he refers to the 3-4 dorsal segments, an area a little lower than the aortic pain of angina (lowest cervical and 1st and 2nd thoracic ?). Tenderness of the left sterno-mastoid and trapezius he refers to the paths of the second and third cervical. Certain cases of sudden coronary embolism attended with pain I shall discuss presently ; in them the pain may be intense, but more continuous, and in seat often epigastric, or truly " precordial." Now if we admit, as I think every clinical observer will be ready to do, that such is our everyday experience of heart pain, as in mitral disease, or in ill-compensated aortic regurgitation without disease of the aorta, we may use this feature as a point of comparison with other pains of cardio-arterial origin. The following patient, brought to me by Dr. Alexander of Walthamstow, is a very interesting example of what I think was " pain at the heart," as contrasted with the pain of angina pectoris (pp. 387 and 430) :

Male, æt. 64 ; for about two years had complained of pain definitely in the cardiac area, rather depressing than severe. When he is sitting perfectly still it disappears ; when he moves it returns. For instance, on walking upstairs and undressing it appears, with some dyspnea, and continues ; when he is settled in bed these symptoms pass off. There is no peculiar dread nor arrest, he can go forward ; but, as to walk up a hill brings on the pain and dyspnea, he is fain to stop for relief. It does not extend nor radiate. Some emphysema was present ; but the heart could be mapped out to 1 cm. beyond the mid-clavicular line. The sounds presented a somewhat laboured systole, and a ringing second aortic sound. Radial pulse thrusting and sustained ; artery fair for age. The systolic pressure indicated at first well over 200 mm., but when every means of quietude had been taken—including emptying the bladder—the reading gradually fell to 180.

Such a pain, probably of cardiac origin, mimics " the heartache of melancholia " (Robert Jones), or of other " disheartened " people ; though this semblance may be only superficial. However, such is the pain of the overdone heart, as seen in Dr. Alexander's patient and in and out of hospitals daily. In this case the heart was labouring under high blood pressures, and the pain was associated not with arrest of respiration

but with dyspnea. This kind of pain is not paroxysmal, and it does not radiate. A grip or even "squeeze at the heart" is occasionally described by such patients, but not comparable with the vice-like compression of the thorax in angina. Sometimes, it is true, the pain of angina also may be continuous and wearing, but only I think between times, in phases of alternation with acute attacks. Dr. Norman Moore[1] says of the more equivocal pain that if cardiac, even if not confined to one spot about the cardiac area, nay, even if "very severe spasmodic pain is also sometimes felt, which might be mistaken for angina pectoris," yet if watched it will be seen to be different in that it is more continuous, lasts a much longer time, often many hours or days, and is not attended with any sense of impending death. In the following case from my own notes we find these two kinds of pain interestingly contrasted.

Male, sent to me by Mr. James of Biggleswade. Burly, rather obese man of 63. Had lived freely. Pain in the chest two months, especially after meals and on exertion. It had two seats of origin, under the left breast—"cardiac region," and about mid-sternum, but a little to the right of the bone. Both pains aggravated by exertion, but the patient insists on a distinction between them. The sternal pain was paroxysmal, radiating down left arm chiefly; when worse passed also to the right, along inner aspects, not beyond the wrist. The pain in the region of the heart was a continuous, weary and depressing local ache, not severe. It was often tedious of an evening when he was quite at rest, and would grow worse when the heart became irregular. No dyspnea. No dread. Radials not very thick, but pressure obviously very high, a thrusting pulse. The systolic pressure had reached 200 when an accident happened to my manometer. Chest too fat for precise examination, but enlargement of heart evident, and pulse hard in carotids and subclavians. Faint systolic aortic murmur, second aortic sound "tinny." Urine normal. No casts, no albumen. Sp. gr. 1025. The diagnosis given was that the heart was painfully labouring in Hyperpiesis, and the aorta severely strained, perhaps to the degree of a subacute aortitis.

This man had both angina pectoris and heart disease, each with its several and different pain. Now I must reiterate that in the pain of angina mitral regurgitation is, generally speaking,

[1] Moore, N., *Luml. Lect.*, 1909.

not an aggravation but, as Walshe and other clinical students, especially Broadbent and Musser,[1] have proved, a palliative. To quote further from Musser; he stated (among other conclusions not relevant): "that when dilatation of the heart supervenes in a patient, the subject of angina pectoris, the subjective symptoms may subside; that angina pectoris may occur in a patient who had dilatation of the heart when the dilatation is removed by treatment." The relief is generally attributed to a coincident fall of aortic pressure; I have said that my experience is to the same effect.

That in mitral disease angina is curiously rare is the testimony of perhaps all observers. But now we have to meet certain exceptions to this maxim, marginal instances, rare but signal; these I propose to discuss with certain other lesions attended with anginiform pains which may however be directly of cardiac origin. These instances, on an impartial view of the facts, I cannot ignore, although they may have to wait a while for their true interpretation; they are instances, I frankly admit, of angina, clinically unquestionable, occurring with heart lesion, and apparently caused by it. By their rarity, and the prepossessions of their observers, these cases have too often escaped a thorough verification; at present however they do point to the possibility of an angina of directly cardiac origin. It is rather remarkable that it should fall to me to justify the occasional use of the term "primary cardiac angina" so commonly, but for the most part so erroneously, in the mouths of our brethren. Regarded on their cardiac aspect these rare cases seem to fall into three classes:

A. Those associated with mitral disease and especially with mitral stenosis;

B. Those associated with thrombosis, aneurysm, or rupture of the ventricle, often with a focal pericarditis.

C. Those associated with pericarditis.

Anginal Pain with Mitral Disease.—In taking a large survey of the subject before us, on the one hand we have seen, in the sphere of the phenomena not directly associated with disturbance of the heart named angina pectoris, some which can be explained by, and are correlated with, lesion wholly con-

[1] Musser, J. H., "Angina Pectoris," *Amer. Journ. Med. Sciences*, Sept. 1897.

fined to the aorta; on the other hand, looking, as broadly, at the phenomena of disease of the heart, we see some pains directly associated with cardiac disease, but as a rule they are not attended with pain of the mode of angina. Viewed in this large way the contrast is so striking we may wonder that its significance has not commanded more attention. But this is not all.

A. In the only four cases, which I can remember, of association of mitral disease with angina no necropsy was obtained; I had half forgotten them therefore, supposing them to be of complex origin; possibly associated with aortic atheroma. Still occasionally I read or heard of other cases of the same association; and Dr. Beddard, who also had seen a few such instances, more definite than mine, kindly recalled my attention to this side of the subject. My four cases were as follows:

The first case was in a certain Mr. F., who was staying for some weeks at the house of his son, Dr. F., who had kept a careful eye on his father's symptoms. Mr. F. was an old man with obvious atheromatous disease of the arteries, and no doubt of the aorta; and symptoms of cardiac disorder, of which I have no precise note, had for some weeks given his son no little anxiety. However, the patient was said to have had no mitral murmur until a certain day, when he was seized with a violent attack of "angina pectoris," and the agony of it persisted so grievously that death seemed imminent. Now, so far as I remember, the symptoms, save that the pain seemed more continuous, resembled those of ordinary angina, with two exceptions; the first exception was the presence of urgent dyspnea with cyanosis; the second was the site of the pain, which in this case was seated no doubt in the cardiac and epigastric area; the patient pressed his hand on the chest below the nipple, and over the sixth rib. A high-pitched murmur, apparently of mitral regurgitation, was audible, and this, as I was assured, had appeared with the first seizure.

We guessed that some rupture of decayed structures of the wall about the valve had occurred; or a thrombosis of a large coronary branch. This was my opinion, the mitral murmur being but an event of some myocardial collapse. I may add by the way that, although the agony could not but alarm the hardiest patient, yet there was not in this case the peculiar omen of death. I find no memorandum about pericardial

friction (p. 461). Of the second case I have no notes made at the time, but I recall that, with the appearance of a mitral regurgitant murmur, anginal pain radiating from the heart's apex set in, but here also with dyspnea and cyanosis. As in both these cases, which seemed contrary to the maxim of relief of angina by mitral regurgitation, there was old standing and severe cardio-arterial disease, they cannot be taken as crucial; especially in the lack of necropsy. The third was more definite, though also without necropsy.

Man, æt. 52; seen January 1904 with Mr. Agnew of Bishop's Stortford. A vigorous, burly, fresh-complexioned man, in his youth an athlete, but latterly stout, and out of condition. Temperament impulsive; health excellent. A few days before had an altercation with a half-drunken workman; a wrestle took place, and the man was "chucked out." A little tremulous, he returned to his desk, and in a few minutes pain of great intensity set in at the chest; it began one inch to the left of the xiphoid, and passed up to the right axillary line. No pain about the apex. Mr. Agnew saw him very soon, and then were there no morbid physical signs. The pain was so intense that he had to be kept under morphia; it was continuous, and never passed into either arm. He was kept at absolute rest, and in two days the pain subsided; when I saw him then he said that while in bed "he was all right." I found the heart a little dilated to the right; the veins of the neck were not swollen, nor the liver, but there was extensive coarse crepitation over the backs of both lungs. Also a loud systolic murmur was audible at the apex (this appeared about seven hours after the attack began); it was audible also behind under the scapula, and at the xiphoid was very intense and superficial, as if tricuspid; probably both the mitral and tricuspid valves were leaking. The pulmonary second sound was accentuated. The pulse was small and apparently of very low pressure, rate about 120-130; when first seen it was 140. He died, but no necropsy was obtainable. This pain, as I have remarked concerning some cases of coronary thrombosis, etc., if akin to that of angina, scarcely behaved like it.

The fourth case was in a girl of 11, the subject of mitral stenosis after rheumatic fever. She suffered from attacks to which the title of angina pectoris could not be refused.

Other recorded attacks of angina with mitral stenosis in young subjects, some in cases verified by necropsy, are more distinctive. Schmoll records such a case of angina with mitral

stenosis and regurgitation, in a girl, æt. 21.[1] There was no pain in the arm, but a paresis of the limb. Dr. Knott has published a similar case.[2] Dr. Samways[3] in getting out a large number of mitral stenosis necropsies at Guy's Hospital, found there had been pericarditis in one-third of them! Dr. Graham Steell[4] has published two "typical" cases, and stated that "the coronary arteries and orifices were perfectly sound." Both were in women, of the ages 22 and 24, in whom atherosclerosis was presumably absent. The signs were those of mitral stenosis, and the symptoms, similar in both, were pain strictly at and round the heart, a dragging pain rather than a sense of thoracic constriction, but paroxysmal, and radiating thence, shooting and tingling to the left shoulder and arm, and left side of neck and face. The attacks usually occurred at 7–9 P.M. and were preceded by fits of dyspnea. After the attack the spine was tender in the region of 1–3 dorsal vertebræ, and there was tenderness in other areas in front. The dyspnea subsided with the pain. The sphygmograph indicated no variation of curve during the attacks. He adds elsewhere the pointed reflection that death in these cases does not result from the angina, but in the ordinary course of valvular disease.

Dr. Beddard was good enough to report to me (in September 25, 1909), two such cases among his outpatients; in one of them, a woman, the angina was of such intense severity that the distress could be palliated only by chloroform inhalation. Venesection gave no relief. The attacks in her were prone to recur when the general symptoms were worse, but also at moments of excitement, exertion, chill, and so forth. Furthermore, Dr. Andrew Elliott was good enough to send me written reports of two cases, in both of which however the association was probably apparent only.

I. Female, æt. 48, who had had rheumatic fever ten years before, and now presents symptoms and signs of mitral stenosis, suffers from very severe typical attacks of angina, with angor, passing at times

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[1] Schmoll, *Münch. med. Wochenschr.*, Oct. 8, 1907.
[2] Knott, *Amer. Med.* vol. viii., 1904.
[3] Samways, D. W., *Brit. Med. Journ.*, Feb. 6, 1898.
[4] Steell, Graham, *Lancet*, Nov. 21, 1903; and Dec. 9, 1911. Also *Index of Treatment* (*loc. cit.*).

into the status anginosus. At her worst she was often relieved by venesection, but was chiefly benefited by nitrites and morphia.

Now in this case the angina may well have arisen from the aorta, for Dr. Elliott noted a diastolic murmur, with some aortic characters, heard faintly and occasionally down the sternum. However, a necropsy was obtained, and in fact "atheroma of the first degree was found at the bases of the aortic cusps, and the aortic orifice was slightly dilated. "*The coronaries were normal.*"

II. The interest of the second case lay likewise in the association of mitral stenosis (in a woman, æt. 54) with anginoid seizures; but these, after some watching, he discerned to be "spurious." They were "spontaneous" and prolonged; the patient was restless and excited and highly emotional; there was no true angor. Moreover, under treatment upon this diagnosis, the anginoid features of the case subsided.

But other cases of genuine angina, supervening upon mitral stenosis, have been recorded here and there; for instance by Ortner,[1] and by Dr. Otto May (two cases of mitral stenosis in women æt. 31 and 45 after rheumatic fever). Dr. May suggests, as Dr. Beddard had suggested to me, that the cause of the angina might lie in strain of the pulmonary artery, probably with the atheromatous injury of the vessel often found in mitral stenosis. If it be so, disease on the right side of the heart does not determine a dextral radiation (p. 301). Ortner endeavours to discriminate between the characters of the pain and symptoms in these valvular cases, and in "typical" angina; but his differences are vague: in many of these stenosis cases the radiations have been quite typical (not even "confined to the depth of the chest"), and the symptoms have differed only in so far as they were blended with those of the valvular lesion. I am disposed, therefore, to wait a while until by careful autopsy, with our eyes open to this part of the problem, we can ascertain whether or not in these exceptional cases of mitral stenosis with angina the aorta, outside as well as within, is intact. For, as the left auricle embraces the root of the aorta, a subinflammatory process may readily extend from the one member to the other; or, as in Elliott's Case I., in Ortner's, and in both of Steell's cases,

[1] Ortner, *loc. cit.* pp. 14 and 24.

both heart and aorta may have been infected by the rheumatic poison.

It is known to students of heart disease that in mitral stenosis palsy of the left recurrent nerve is no infrequent symptom ;[1] but we are still more familiar with it in diseases of the aorta in which angina pectoris also is not infrequent. Accordingly, Boinet[2] suggests that, without compression of the nerve by dislocated parts or enlarged glands, a periaortitis may account for this recurrent palsy ; indeed in three of the reported cases chronic adhesive pericarditis is mentioned, and in one of Herrick's cases[3] the left recurrent was imbedded in a mass of cicatricial tissue. Fetterolf and Norris conclude that, by distortion of the parts in mitral stenosis, the nerve is indeed compressed between the dilated left pulmonary artery and the aorta ; but, considering the softness of the parts, and the normal flattening of the nerve against the aorta, they think that something more is required to account for the effect, and suppose from the evidence that the nerve is palsied by a neuritis propagated from the pericarditic, the glandular, or other local inflammations which are found in some of these cases. Thus the carotid pulses may be unequal ; and Muktedir[4] describes two cases of mitral stenosis in each of which the voice altered, and palsy of the left vocal chord was demonstrated. The right pupil was dilated in both. Kraus thinks the recurrent nerve cannot be pinched by the auricle, but is dragged and atrophied by the downward swing of the parts ; but if so should not this effect be more common ? Lian and Macorelles,[5] reviewing this subject, coincide in opinion with Fetterolf and Norris. They quote a case in which they demonstrated a corresponding local chronic mediastinitis ; and from another source one in which after death a perivascular inflammation was found. Now if in mitral stenosis such a process

[1] For a careful study of this event see Fetterolf and Norris, *Amer. Journ. of Med. Sciences*, May 1911 ; and Cohn, G. (of Konigsberg), "Linkseitige Rekurrenslähmung in folge von Mitralstenose," *Arch. für Laryngol.* Bd. xxiv. H. 1, 1910.

[2] Quoted by Fetterolf and Norris, *loc. cit.*

[3] Herrick, *Tr. Ass. Am. Physicians*, vol xxvii., 1912, narrates several cases with necropsies. Some had had anginal attacks, some not.

[4] Muktedir, *Deutsche med. Wochenschr.*, 1911, No. 11.

[5] Lian et Macorelles, *Arch. des mal. du cœur*, juin 1913.

may arise, it may account also for the occasional coincidence of angina pectoris, as likewise engendered by a periaortic inflammation. All we can say as yet is that mitral stenosis may be associated with irritative lesions round about. As I have said elsewhere, we have been too content with a static view of valvular diseases, and to regard the morbid process as confined to the parts of the valve and orifice. The observations of Carey Coombs, of the Leipzig school, and of others tell us that these processes are more subtle and pervasive. Dr. Hare of Philadelphia has reminded us that Mahomed demonstrated how prone was what we now call partial heart block to ensue in mitral stenosis; an observation which was verified by Galabin. Recently Mackenzie, Lewis, Herringham, Hay, and others have illustrated this course of events; and led us to see how chronic inflammation may be propagated around the mitral area, and, as I suggest, may gather occasionally about the root of the aorta, and be revealed by angina. In malignant endocarditis, again, a complexity of this kind would be intelligible.

Professor Allan,[1] whom I have quoted elsewhere (p. 172), in his long series of necropsies in cardioarterial cases, includes instances of stenosis due to syphilitic thickening of the fibrous rings about the valvular orifices. These may have been cases of periaortic syphilis.

From these instances at least two points of importance emerge; namely, that Ortner in these autopsies found the coronary arteries quite normal; and, secondly, that if it be true that the subjects of mitral stenosis may, very rarely, be prone to angina, then angina does not arise from straining of the left ventricle, which usually in these cases is underloaded. It is the right ventricle which labours under excessive stresses. Therefore, in the absence of more post-mortem evidence, and especially of closer examinations of the parts around the aorta, we must suppose that in these, as in the rare instances of angina with mitral regurgitation, some other factor was concerned of which we have no information, but which we may guess at.

B. I have now to appreciate some similar evidence that angina pectoris, or pains not unlike it, may be caused by aneurysm and rupture of the heart itself, or by *sudden*

[1] Allan, *Intercolon. Med. Journ. of Australia*, March 1909.

coronary thrombosis, or very rarely, *embolism* (p. 370); now if this be so, we may again be constrained to admit the possibility of anginal pain originating about the heart. In common report we hear of two modes of sudden death in angina; the one a sudden snapping of the thread in an ordinary paroxysm, the other in a no less sudden, but also violent and protracted, seizure which is the first and the last. The patient, who probably has already shown signs of decay, is killed by a long and terrible anginal agony. Two such cases have occurred in the last twelve months in my circle of friends. These are cases, I believe, of coronary thrombosis. We must remember that in the excitement of a fulminant and mortal seizure, precision of notes is not to be expected; and it may be that in some cases the name Angina is hastily attributed to any violent pain in the chest, whatever the associated symptoms. But in many of them it is true that a substernal pain of intense agony, with radiation to the left arm, is recorded definitely enough. Dr. Parkes Weber has kindly directed me to the records of many cases [1] in which a sudden block of a large coronary branch was attended with severe local pain. From these cases, and some others, I find the distress was associated with a pain, substernal, epigastric, or submammary, more continuous, it is true, than ordinary attacks of angina pectoris, but apparently of the same nature (p. 368). In all of them however the agonizing dyspnea and cardiac failure pointed to a causation different, if not wholly different. Morgagni says he does not know how to make an end of writing on these cases (aneurysm of left ventricle, etc.). In older days these, and such, sudden painful deaths were attributed to poison; but Morgagni emphasises lues as a main cause of such explosions in the arteries and heart (*vide* Ep. XVI.). A clinical difference is illustrated however by a case of Dr. Morison's,[2] in a man æt. 59, the subject of a fatal thrombosis of the right coronary artery. During the period of his survival (about 24

[1] The principal are Church's case, *St. Bart's. Reports*, 1896, vol. xxxii. p. 17; Korczynski's, Abstract in *Jahresb. ges. Med.*, Berlin, 1887, vol. ii. p. 219; Hektoen's, *Medical News*, Philadelphia, Aug. 20, 1892, p. 210; A. Morison's, *Harveian Soc.*, Nov. 25, 1909; Garrod's, *Proc. Roy. Soc. Med.*, April 1908 (the same reference also to Parkes Weber); J. Mackenzie's, *Heart Disease*, p. 44, 1908. Cases referred to by His and Beitz, *Gesellsch. d. Charité-Arzte*, Berlin, Jan. 6, 1910, are reported in *Berl. klin. Wochenschr.*, 1910, xlvii. p. 645.

[2] Morison, A., *Lancet*, Jan. 8, 1910.

hours), the kind of pain, and the collapse, suggested perforation of gastric ulcer; a behaviour not quite characteristic of the pain of angina, even in its most importunate or tenacious assaults. The same difference is to be found in the notes of a case of coronary thrombosis most kindly furnished to me by Dr. G. E. Higgens of Hornsey Road, N.

Man, æt. 47, seized on a Saturday about mid-day—not during exertion—with sudden pain about the lower end of the sternum; vomited frequently, and complained of dimness of sight. He became collapsed; the pulse ceased in the right wrist, in the left was feeble, rapid, and intermittent. He died on the Sunday evening. His mind was unclouded till the end. During this last 30 or 40 hours of life the pain was extremely severe and continuous, so much so as scarcely to be assuaged by morphia; it was slightly relieved by turning into the semiprone position, so as to maintain pressure upon the chest. The seat of it was the lower sternal and left hypochondriac region. He also suffered (and this is a fundamental distinction) from extreme dyspnea. An interesting fact in this case was the accession on the Sunday afternoon of cramp in the left hand, the fingers being semiflexed and inextensible. The autopsy (at the G. N. Central Hospital) by Dr. E. H. Shaw revealed only slight atheromatous changes in the coronary arteries; but the right vessel, for the first $1\frac{1}{2}$ in. large and empty, was at this farther point completely obstructed by an irregular red clot extending almost to the junction of the transverse and posterior descending branches. There was a small narrow clot in the transverse branch. The heart was rather large and fatty, especially the right ventricle.

The patient was seen in life by many observers, who variously thought his case to be one of angina, or of rupture of the heart, of a coronary artery, of an aneurysm, or of a visceral ulcer.

There is no reason in the nature of things why anginal pain should not be as continuous as pain may be; in origin it is not rhythmical but contingent; it does not arise from synchronising of multiple oscillations, peripheral and central, nor on inherent nervous capacities but, determined by contingency, passes with it. Whatever the contingency, be it tide of blood pressure, local injury, or other urgent condition, if the cause be continuous, so will the pain be, so far that is as the sensorium can take it up; and, until exhausted, it can take up ten stimuli per second: a less frequency than this is virtual continuity, the shorter negative phases we fail to apprehend. What distinguishes these em-

bolisms then from angina of the ordinary form is not the kind of pain—for this, if it be more about the heart, may be substernal, and quite anginal in seat—but other variables; the flutter and lability of the heart, the rapid, irregular, and failing pulse, the waning cardiac sounds, the dyspnea, the cyanosis. Hochhaus,[1] in a description of four cases of sudden stoppage of a coronary vessel, points out also that the anginoid pain is more continuous than in typical angina pectoris; and that the pain fades into the background as the heart gives way. I shall quote from this paper again presently. Angina is not always present. Sudden embolism of a renal artery sometimes causes pain in the loin, sometimes not. Embolism of the splenic artery, no very rare event, has in my recollection never caused notable pain, presumably because the spleen is a loosely knit and distensible organ. The conditions of tension in these several cases are various. Kauffmann (in the 6th edition of his *Lehrbuch*) says that anginal pain is caused only when a main coronary trunk, right or left, is suddenly jammed. In three cases of sudden thrombosis, reported at Kiew,[2] the pain, at times radiating, resembled angina pectoris very closely; but "the principal objective signs were of 'meiopragia cordis'—disappearing impulse and diminishing sounds"; the prominent symptoms dyspnea and heart failure. The chief clinical distinction to remember in coronary thrombosis is that, if in a large branch, angina may be present but is inconstant; while dyspnea and rapid fall of blood pressure, symptoms which in ordinary angina do not occur, are constant.

The literature of these cases, if we omit the imperfect records, is not large. Tiedemann opened the subject in 1843; but the earlier writers, ignoring symptoms, or taking refuge in the "neuroses," hyperæsthesia plexus cardiaci, and such guesses of von Romberg, Bamberger and others, confined themselves to pathological anatomy. Von Leyden in an able paper[3] pointed out that angina is not an obligate clinical symptom of this thrombosis, and, though occasionally typical enough, is often indefinite or aberrant, and associated with dyspnea, dropsy,[4]

[1] Hochhaus, *Deutsche med. Wochenschr.*, 1911.

[2] Obrastzow and Strachesko, "Thrombosis d. Kor.-Arterien d. Herzens," *Zeitschr. f. klin. Med.* Bd. lxxi., 1910.

[3] Von Leyden, *Zeitschr. f. klin. Med.*, 1884.

[4] See also Panum, *Virch. Arch.* Bd. xxv., as an earlier observer of the subject.

etc., especially in the milder cases with temporary survival; but that in all sudden blocks of large branches dyspnea, collapse, and heart failure occurred. Von Ziemssen wrote later to the same effect.[1] With Morgagni's case [2] no symptoms are recorded. Mayer points out that, as it may be associated with thrombosis, so angina may be caused by aneurysm of a coronary artery.[3] Obrastzow and Strachesko, of Kiew, record angina in three of their cases, thus:

Man, æt. 49. Angina; block of left coronary; sclerosed aorta; myomalakia and patch of pericarditis. In Case II. thrombosis: no angina; intense dyspnea; rupture. In Case III. thrombosis: angina; (but as the aorta was much diseased, and with "atheromatous ulcers," the pain may have originated there).

In most of these sudden coronary blocks the angina appears in the form of "status anginosus." English observers have been more vague in correlating anatomy with symptoms; and both English and foreign observers have too often forgotten to record the condition of the thoracic aorta. In a recent article Herrick of Chicago [4] divides the cases into four classes: (1) Sudden death, in pain; (2) angina, with death in a few minutes; (3) non-mortal attacks, with slight angina (obstruction of a small branch); (4) graver forms, mortal after some longer period; of these he gives six cases, and one necropsy: Symptoms; angina often severe, feeble quick pulse (sometimes retarded), feeble cardiac sounds, dyspnea, cyanosis, pulmonary congestion. In the necropsies pericarditis.

Dr. Parkes Weber has published [5] one case of angina pectoris in which thrombosis of the right vessel was found; the obstruction however was an old one, and, as the reporter pointed out, might have existed before the angina began; for new blood vessels had formed about the seat of the plug. The patient was a diseased man of drunken habits, and "there was also atheroma of the aorta." Still after all there remain not a

[1] Von Ziemssen, *Berl. klin. Wochenschr.*, 1901, S. 253.
[2] Morgagni, *De sed.* l. ii., Ep. 24, 16.
[3] Mayer, *Deutsche Arch. f. klin. Med.* Bd. xliii.
[4] Herrick, Summary in *Arch. des mal. du cœur*, mars 1913. See also *Gaz. des hôp. de Paris*, Séance 29, janvier 1909.
[5] Parkes Weber, *Trans. Pathol. Soc.*, London, 1896.

few striking cases of the coronary thrombosis and myomalacia, not all of them attended with patches of pericarditis, in which nevertheless attacks of pain occurred to which the name of angina can hardly be denied. In respect of the pains and their degrees, the time element, a gradual or a sudden occlusion, makes much, perhaps all the difference. I will consider the bearing of these cases on my hypothesis of angina after dealing with angina and pericarditis.

C. *Pericarditis and Angina.*—In a case of heart rupture with angina, recorded in an interesting article by Dr. Sidney Phillips,[1] a patch of pericarditis was found over the seat of the rupture. There were two attacks of angina, the second mortal. The pain of pericarditis, often stabbing or boring like that of other serous membranes, is in ordinary experience referred to the "cardiac area," not in the careless use common in descriptions of angina pectoris, but in a proper sense of this phrase. That there are cases of pericarditis—somewhat rare, but significant—in which the pain is like that of angina pectoris, has been known for some years to careful observers, such as Sibson,[2] Byrom Bramwell, Andral,[3] Peter,[4] and others.[5] They might have spoken more plainly, and said that pericarditis is occasionally associated with angina pectoris, for an analysis of these exceptional cases may help us to interpret this stealthy disease. Andral writes: "Ces douleurs rappellent *à s'y méprendre* celles de l'angine de poitrine" (italics mine). In the earlier records, as for instance in one of Fothergill's necropsies,[6] our forefathers were puzzled by the discovery in some cases of angina of pericardial, and even of pleural, inflammations. The remarkable case of angina recorded by Haygarth of Chester[7] proved on autopsy to be one of mediastinal suppurative inflammation, apparently involving the base of the heart and the great vessels.

[1] Phillips, S., *Lancet*, January 28, 1897.

[2] Sibson, A., Reynolds' *System*, 1877.

[3] Andral, *Clinique méd.* tome iii., 1834.

[4] Peter, *Leçons de clinique médicale*, 1880, tome i. p. 419. (Seizures radiating into the left arm.)

[5] See also Steell, Graham, on "Pericarditis" and "Angina," *Med. Chron.*, Nov. 1913; a paper which came into my hands too late for this essay.

[6] Fothergill, *Lond. Med. Obs.* vol. v. See also Haygarth's case, *Med. Trans.* vol. iii.

[7] Haygarth, in vol. iii. of the *Med. Trans.*, 1772.

Dr. Graham Steell writes:[1] "The combination of pericardial friction sound with angina pectoris is seen often enough to leave an impression on one, and is occasionally noted by others, as by F. Roberts in Allbutt and Rolleston's *System*, vol. vi. p. 47, by Gibson in *Reynolds*, and by Walshe." Steell relates four cases. Case I. illustrates the fugitive course of the sound, although the pericarditis proceeds; the third illustrates "exactly typical angina" with pericardial friction, ending in recovery. In No. 4, of pericarditis, "the diagnosis of angina was (clinically) clearly established." Gallavardin[2] says under Pericarditis: "Its pains can in certain cases put on the character of angina (Andral, Sibson, Peter) . . . and the patient may suffer from the (peculiar) angoisse."

Now as to the facts; in anæsthetised animals to pinch or stretch the pericardium sends up the blood pressure (Kidd, Mathison), an effect lessened but not abolished by section of the phrenic; the pericardium appears to have a further sensory supply. The pain of pericarditis may extend from the cardiac area, and, as we shall see, may perhaps reach the left shoulder, even while the inflammation is confined strictly to the cardiac surface. Erasmus Darwin (*loc. cit.*) noticed in pericarditis that the pain might travel to the left shoulder, a feature which he attributed to direct nervous conduction. This possibility I have discussed on another page, and I shall return to it. But my present purpose is to dwell more definitely upon angina pectoris, in the strictest sense of the word, as an effect of pericarditis. Sansom wrote, concerning pain in heart diseases, "Pericarditis may be painless; but usually there is considerable distress, which is referred to the heart, but it may extend to the left shoulder and arm. . . . *In rare cases it is paroxysmal, and has the characters of angina pectoris*" (italics mine). Sturges,[3] in one of his cases of pericarditis, a girl, æt. 5, reports that "she suffered a sort of anginal attack." Dr. Samuel West[4] writes on pericarditis, "Cardiac pain of the acute kind at all resembling angina is very rare, and is met with only when there is acute cardiac dilatation, but by no means always then." The qualification seems to weaken

[1] Steell, G., *Med. Chron.*, Nov. 1913.
[2] Gallavardin, *Mal. du cœur*, 1908, p. 212.
[3] Sturges, O., "Lumleian Lectures," *Brit. Med. Journ.*, March 10, 1894.
[4] West, S., *Brit. Med. Journ.*, Oct. 26, 1907.

his opinion of the cause (p. 386); not to mention the difficulty in pericarditis of discerning degrees of cardiac dilatation. Conversely, in no disease indeed is acute dilatation of the heart more common than in acute rheumatism, yet with it " pain resembling angina is very rare " ; there is room then for great hesitation in assuming the cardiac dilatation and the angina to be cause and effect. Dr. James Mackenzie again, in a private letter,[1] kindly invited " my attention to rare cases in which the pain of pericarditis has the same distribution as in angina pectoris." I welcomed this further testimony to facts which had not escaped my attention, and which in my *Cavendish Lecture* (*vide* p. 160) I had already made some endeavour to explain. Dr. Byrom Bramwell [2] also has described a case of angina associated with a pericarditis, which in this instance seems to have been syphilitic, as follows:

Workman, æt. 49, well built and nourished. History of syphilis and alcohol. He was attacked by angina pectoris, but for a day or two nothing abnormal was to be found about the heart; then, however, a distinct pericardial rub became audible. The pericarditis lasted for a fortnight, *after which time the patient made a complete recovery* (italics mine). *N.B.*—An instance, among many, for those who deny recovery in angina. See also Kernig's and Ferrio's cases (pp. 459-460).

Dr. Bramwell confessed that the interpretation of the case remained obscure to him.

Teissier,[3] in cases of the pericarditis of Bright's disease, reports " angoisses rétrosternales quelquefois très vives " ; but with dyspnea also. Barié writes to the same effect.

In his *Morison Lecture* for 1904 (p. 35), Gibson said, " In pericarditis anything approaching the typical symptoms of fully-developed angina pectoris is undoubtedly rare; occasionally however a case presents itself with the characteristic distribution and special features of a true anginous seizure." Why not put it in plainer language, and say that in some rare cases of pericarditis we meet with angina pectoris. Weintraud [4] under " Pericarditis " says that patients often complain (" berichten die Kranken

[1] Dated March 19, 1903.
[2] Bramwell, Byrom, *Diseases of the Heart*, etc., 1889.
[3] Teissier, *Bull. méd.* 12 avril 1911.
[4] Weintraud, in Eulenberg's *Lehrbuch*, ii. 323.

oft") of a severe pain behind the sternum and through to the back; or radiating up to the neck, and sideways into the arms. In its fuller manifestations (he adds) this pain corresponds to typical angina pectoris; so that presumably the slighter but similar manifestations are of the same nature. A short time ago I read in an article by an eminent German physician, whose name has escaped my memory,[1] that the pain of pericarditis ranges from a slight stitch or oppression up to the most intense ("intensive") attacks of angina. Now it is remarkable that all these writers on pericardial angina—so far as I remember, all of them—refrain from the customary tribute to the coronary arteries. Why is this courtesy withheld?

In a paragraph on pain in acute rheumatism of the heart, Weill et Nouriquand[2] say that beside the "simple gêne précordiale," spontaneous or called forth by pressure, less commonly ("moins fréquemment") intense pain radiates beyond the cardiac area, with pallor and anxiety. They point out that these pains are sometimes comparable with those described by Peter and Letulle, and attributed by them, as by Lancereaux and some earlier clinical authors, to neuritis of the cardiac plexus. They allude to the opinion that acute myocarditis may be painful, a speculation into which however they decline to enter; they content themselves with the demur that, "keeping strictly to the exclusive domain of facts," in the necropsies of such cases they have found nearly always a recent, generally slight, pericarditis.

The controversy must be decided by post-mortem evidence, yet, if pericardial angina is rare, autopsy in such cases is still rarer; but it is generally understood that a partial dry pericarditis may occupy the parts where the great vessels leave the heart. In the article on Aortitis, I have alluded to Pawinski's very important evidence, and to Auscher's evidence[3] from a very interesting case of "posterior basic pericarditis," with angina, to indicate that the cause of angina in pericarditis may lie in the extension of the inflammation to that tract or collar, thus invading the periaortic structures.

[1] In the *Deutsche med. Wochenschr.*, May 19, 1910.

[2] Weill et Nouriquand, "Cœur et rheumatisme viscéral," *Paris médical*, 1er juillet 1911.

[3] Auscher, *Bull. Soc. Anat. Paris*, février 1896.

Auscher's case was as follows:

Male, æt. 61. Of sober habits; never suffered from syphilis or rheumatic fever. No tobacco. For a few months previously had been subject to severe and typical angina pectoris; he described the pain as of a red hot bar squeezing the chest violently, an interior pain. One of the attacks ended in syncope. After admission to the hospital he had had one attack, diagnosed clearly as angina pectoris; and a distressing sense of oppression was often felt. A double murmur was heard about the base of the heart which was attributed to aortic disease with regurgitation. Nine months after discharge he was readmitted for rapid phthisis, and died. (As no farther mention is made of it, the angina presumably had ceased.) At the necropsy the heart was found perfectly healthy, the aortic and other valves normal, and the aorta "supple and elastic." "Les orifices des coronaires étaient irréprochables," and these arteries were "sur toute leur longueur souple et perméable." Sections showed also that the walls, both as to vessels and muscular tissue, were normal. The integrity of all these parts was "nettement demontré." Then what was found? What was the explanation of the angina? Around the base of the heart and the root of the great vessels, and on the corresponding portion of the parietal pericardium, were definite signs of old chronic pericarditis, and upon these areas was an opaque false membrane. *This is a crucial instance of angina with sound heart and sound coronaries, but with inflamed aortic investment.*

Pawinski's [1] evidence again is that these latent inflammations of the pericardium, with more or less restricted highly fibrinous exudates, may attack this part of the sac, the part which invests the roots of the great vessels. He records eight cases of his own which were marked by anginal pain of intense degree, especially at the onset. In some cases in which the pain was most violent, the pulse began suddenly to fail—"a sudden fall of the heart's action ('Herzthätigkeit'), with a weak and irregular pulse" (vagus symptoms?). Indeed these stenocardial effects he relies upon as symptoms of this one of the latent forms of pericarditis, suggesting that neuritis of the plexus cardiacus may be the most important source ("Quelle") of "stenocardia." He denies that an explanation can be found in coronary disease and myocardial anæmia, for in his cases it was the pericarditis which called forth the pains peculiar to angina—"die Angina Pectoris

[1] Pawinski, *Deutsche Arch. f. klin. Med.* Bd. lviii., 1897.

eigenthümliche Schmerzen"—without the coronary arteries being involved, or in any way concerned; as we saw in the syphilitic case recorded by Merklen (p. 375). In one of these, a case of Pericarditis sicca, Pawinski noted physical signs of dilatation of the ascending aorta. Now, had such attacks occurred in old persons with diseased arteries, they might well have proved mortal by inhibition; however, in all Pawinski's mortal cases death was due to other causes. I may here refer to Perez' case without repeating it. Von Romberg[1] writes likewise: "Sometimes (in pericarditis) the anxiety is greater, and is accompanied by pain radiating into the neck and arms, especially the left arm, resembling angina pectoris; unpleasant symptoms which may precede the more objective phenomena by a day or two."

As further evidence of the same kind, I would cite also an interesting paper by Luigi Ferrio,[2] of Professor Bozzolo's clinic. This paper I had procured for other reasons, but in it I noted also some remarks, apt in this place, on the propagation by continuity of inflammation to the aortic valve from that part of the pericardium which encloses the roots of the great vessels. As probably in the following case:

J. M., æt. 17. Acute rheumatism; ten days after admission, a recrudescence of fever with pericarditis and intense pain, "allo sterno e nelli parti adjacenti"; soon afterwards the aortic valve was attacked. Paracentesis pericardii, withdrawal of turbid serum with flocculent fibrin. Relief, and ultimate recovery.

Boinet[3] likewise says, "Acute or subacute aortitis also may result, rarely it is true, by propagation of inflammation by contiguity in pericarditis and in endocarditis; especially in rheumatism.

Pawinski says that the angina does not occur with pericarditis in other parts of the sac, an opinion which, as we shall see, is open to some doubt, or qualification. I may note, notwithstanding, that it is in pericarditis about the origin of the aorta that angina is most apt to appear. I note also that irritation of the pericardium sets up vagus inhibition (extrasystoles, etc.). Cocainise the pericardial area and the vagus ceases to respond.

[1] Von Romberg, *Krankht. d. Herzens*, 1906, p. 372.
[2] Ferrio, L., *Gaz. degli osped.*, Sept. 24, 1905.
[3] Boinet, in Brouardel and Gilbert's *System*.

Let us now turn to the reverse of these events, to a possible propagation of subacute inflammation from the aortic area to the pericardium: five such cases are recorded by Kernig;[1] of these I will give a scanty summary.

I. An elderly general officer, subject to angina. After a severe attack his temperature rose, and a pericardial rub was heard at the base of the heart. II. Physician, æt. 47. After great effort, severe attack of angina ensued, with rise of temperature and pericardial friction. Symptoms passed off in ten days. Convalescence satisfactory, and return to full work. III. Army officer, æt. 56. Severe first attack of angina (lasted 6 hours). Two or three days later some rise of temperature and a pericardial rub. Three weeks in bed. On rising from bed, sudden death. IV. Merchant. Æt. 68. Angina. Then a bad attack lasting 24 hours. Two days later rise of temperature and pericardial rub. Feverish for a fortnight; then complete recovery from all ailment. V. A similar case narrated to the author by a colleague.

At the Pesth Congress in 1909, and again, I think, at the Berlin Congress of 1912, Professor Sternberg of Vienna read an interesting paper on this aspect of the subject, entitled "Pericarditis epistenocardiaca."[2] In opposition to Pawinski, Sternberg and Kernig insisted, rather topsy-turvily as it seems to me, that the anginous symptoms came first, and that the pericarditis followed them; *i.e.* a bacterial or other infection attacking the myocardium causes the angina, and the pericarditis is but an epiphenomenon. Sternberg produced two cases, with a necropsy in one of them. This was of a man of 47 (age at death 51) with a long story of syphilis in self and wife. He had nasal disease with asthma, and for some years symptoms of disease of the arteries and heart. On April 26th he had an attack of angina. On May 23, 1906, occurred a second very severe attack of angina pectoris, and within 24 hours a pericardial rub was heard. Temperature 39·2. His stenocardial attacks seem to have persisted, as a report in 1907 speaks of them as less frequent. After this a period of amendment was obtained, but then a gradual dissolution set in, with much asthma and cardiac failure, amongst which sufferings little is said of angina.

[1] Kernig, *St. Pet. med. Wochenschr.*, 1892, and (with five new cases, but no necropsy) *Berl. klin. Wochenschr.*, 1905. See also Bramwell, B., *Diseases of Heart and Arteries*, 1889.

[2] Protocol, published *Wiener med. Wochenschr.*, No. 1, 1910.

Of the necropsy it is sufficient to state that a chronic partial aneurysm of the heart was found associated with old pericarditis; the aorta exhibited the well-known syphilitic destruction, and the coronary arteries also were extensively diseased. So that although the acute seizure attended with pericarditis determined one of the attacks of angina, or coincided with it, yet without it there was quite enough specific mischief in the aorta to account for this event. Upon a case so complex no decisive argument can be founded. The second case was in a man of 74, in whom an attack of pericarditis occurred with angina. He made a good recovery. Sternberg's opinion is, as I have said, that in all such cases the pericarditis follows the anginal seizure, at an interval however so short—a few hours—that from the ear alone it would be impossible to time its onset precisely; in any case not before the tough tunic was stretched by exudation into its interstices.

From a survey of the cases with necropsy, and a reading of the rest by their light, it would seem that the chief features of Sternberg's cases (*loc. cit.* p. 368) are a sudden block of a coronary artery—whether by an embolus or by thrombosis—with Ziegler's myomalakia, and disposition to aneurysmal pouching; upon this rides a superficial pericarditis, as on the pleuritis of a pulmonary infarct. In Sternberg's opinion the formation of aneurysmal dilatation relieved the angina; with it the tension may have yielded. He called the stenocardial (thrombosis with hyperæmia ?) the first stage; the pericarditis marked the second; the third was the latent stage of apparent recovery; and the fourth, the decay of the cardiac muscle. But on current notions the third and fourth stages ought to have been those of the stenocardia.

After the publication of Sternberg's paper appeared that, to which I have alluded, on the same subject by Professor Hochhaus of Cologne,[1] but dwelling more especially on the diagnosis of sudden coronary block (p. 450); he reports instances of such diagnosis during life by Hammer, Curschmann, Obrastzow and others.[2] Hochhaus describes three

[1] Hochhaus, *Deutsche med. Wochenschr.*, Nov. 9, 1911.

[2] See Krehl, *Erkrankungen d. Herzmuskels*, Wien, 1901. Also *Zeitschrift f. klin. Med.* Bd. lxxi. He. 1 and 2, with bibliography.

cases, all with pain of a more or less anginiform character; but as in two of them pericarditis was present as well as aortic disease, and in one of them a history of angina in its ordinary form, I will concentrate attention on his third case. This was of a woman, æt. 69, who was seized with the symptoms which I have indicated, including severe substernal pain—a group of symptoms which, so Hochhaus argues, seems to suffice for diagnosis of this accident. Considerable aortic atheroma was found after death; and the coronary arteries were extensively thrombosed (both with old silting up and several recent acute plugs). Now in this case the pericardium was smooth and bright; so that pericarditis is no necessary mean in thrombotic angina. The myocardium however, besides necrotic changes, presents in these cases, especially about the apex, hæmorrhagic swellings and acute fibrinous exudations into its substance, and these changes in Hochhaus' case were well marked. I have discussed these problems with Professor Hochhaus, and I admit that this instance again, most carefully described, seems to be almost crucial; I can no longer hold that the source of angina pectoris is exclusively aortic, though it is possible that the atheroma of the aorta, long latent, sprang into life and set up angina at this critical moment. No periaortitis is recorded. In these cases then the intense anginiform pain appears to have been generated, not by any extension of inflammation from the pericardium to the root of the aorta—*vera causa* as this is—but in the heart itself. The interpretation of the pain however in my opinion still lies in differential tension of investment, in an engorgement and distension of the affected portion of the pericardium by the sudden hæmorrhagic and fibrinous exudations. This stretching of investment, and perhaps of fibrous attachments, evokes pain in the same way as at the root of the aorta it is evoked by the distension of systole, or by rupture with sudden escape of arterial blood between the coats of the vessel. But, as we might anticipate, the pain is more continuous. It is to be noted that in all (?) these cases there is pain about the heart itself as well as in the sternal area; and that the angina is a feature not, as would usually be assumed, of the chronically enfeebled heart but a transient feature of the initial phase only. Dilatation of a whole ventricle is not thus differential,

is less sudden and is not unnatural; still dilatation is not rarely attended by a sickening ache. I have said that the agony of mesenteric embolism is presumably to be explained in the same way; that is by the tension and drag on the mesentery, which is rich in sensory nerve endings, and by the thrust of the sudden secondary engorgement and effusion.

But nearly all these records are defective in lucid clinical distinction between continuous pain in the cardiac area, which we know does occur in some cases of sudden coronary embolism, and the paroxysmal sterno-brachial pain of angina; also they lack definite delineation of the several areas, the mind being occupied with the ruin of the wall of the ventricle. Moreover in most of these cases, besides the pain, there were many signs of heart disease and heart failure; they suggest that under these conditions a coronary plug and myocardial destruction do not suffice to produce angina, that they are not its cardinal factors; that to set up angina the tough pericardial investment must be suddenly and differentially stretched, by exudation, or by some local surge of the blood. Kauffmann (*loc. cit.*) says indeed that beyond the plug a strong hyperæmia sets in, even to hæmorrhagic with other exudations, sometimes to rupture. Ordinary pericarditis in such subjects, without the factor of aneurysmal pericardial distension—as for instance in Bright's disease,—even with coronary disease thrown in, does not set up angina unless it embrace the root of the aorta. I reiterate that in many cases of angina with pericarditis, as for example in Auscher's well-known case,[1] in some of Pawinski's, and many others, the coronary arteries were intact.

Hampeln[2] has carefully surveyed a long list of cases of rupture of the heart and aorta, and found the characteristic difference between them to be the predominance of anginal pain in the aortic cases; pain in the cardiac cases being often absent, or ill-marked. (See Aortitis, p. 206.) Now we have seen that a rupture of the ascending aorta, stretching the investment of the vessel by the dissecting blood, causes pain analogous to that of periostitis. Dr. Humphry informs me

[1] Auscher, *Bull. de la Soc. de l'Anat. de Paris*, vol. x., fév. 1896 (see p. 458).

[2] Hampeln, *St. Petersburg med. Wochenschr.*, 1899. A paper of which I have seen only abstracts in other journals.

that in a recent case of his own, in a young man in whom after a violent effort symptoms of severe angina pectoris ensued, at the autopsy a clean rupture of the aorta a little above the valve was found, the blood having dissected up the coats. The heart was quite normal, and the aorta, though a congenitally slender vessel, was free from disease. There was no syphilis. There are on record not a few of these splits of the aorta with angina, and in most of them, before autopsy, a diagnosis of angina pectoris was made. D'Antona has published [1] an interesting summary of these sharp-edged ruptures, with two cases of his own; in one of these, in which the pain was intense, the coats were split asunder by the dissecting hæmorrhage. (Similar cases (*e.g.* Pallasse's case, p. 207) and a further discussion of this subject are to be found in the chapter on Aortitis, p. 206.)

In all these rupture cases the adventitia shews itself remarkably tough, and is not torn; it is the last strong guard of the integrity of the vessel. Now these aortic ruptures are analogous to the forcing of the visceral pericardium in the coronary thrombosis of which we have just been speaking.

Aneurysm of a coronary artery seems to arise only in the course of embolism (Kirkes and Wardrop Griffith); [2] if angina be present it would probably depend not upon the aneurysm, which of itself might not cause much tension, but on some associated local condition.

Now if for the moment we consider the facts of the common association of angina pectoris with aortic disease, especially with its acuter inflammations and strains, facts of which we have all this plenty, we must be struck with the convergence of their evidence towards a conviction of their causal dependence. Even in respect of angina arising in the heart itself I have shown that in this area we have been too wilfully bent upon seeing nothing but the coronary arteries or myocardium. In Auscher's case of pericarditis sicca attended by three attacks of severe and typical angina pectoris, when six months later the patient died of phthisis the remains of false membrane were

[1] D'Antona, *Arch. per le sci. med.*, 1911, vol. xxxv. fasc. 4.

[2] Griffith, Wardrop, *Brit. Med. Journ.*, Feb. 22, 1901: with a good list of records.

found on the roughened surfaces of the visceral and parietal pericardium investing the origin of the great vessels; and I repeat the author's declaration "that in spite of the typical angina and parietal pericarditis around the aorta" . . . "l'orifice des coronaires était irréprochable . . ."; that these vessels were beyond reproach "dans toute leur longeur," and on section proved in histological structure to be normal. Is not this crucial? Gallavardin's anxious flutters between loyalty to Huchard and the coronaries and the evidence of his own acuter observation are diverting. In one breath he says with emphasis that angina does not arise from a "coronarite véritable" but nearly always from an aortitis propagated to the orifices of the coronaries; nay, he quotes Bard and Morvan, and under the influence of Josué and myself admits that we must remember "c'est l'aortite qui importe surtout, non la coronarite"; yet he is perpetually harking back to the old notion, and losing his way. He tries to find some controversial refuge in a neuritis.

On the other hand, as I have said already we are all agreed that by sufficient slowness of approach, a kind of tolerance, which may be illustrated from many kinds of disease, may be established. This immunity of whatsoever structures, coronary or myocardial or aortic, if the disease be but slow enough, is well illustrated by the following case, recorded by Dr. Parkes Weber.[1]

Male, æt. 61, syphilis, and gumma of liver. Extensive atheroma of the aorta, which at its root seemed to involve all the coats. At some places the pericardium was adherent, as if by a panarteritis rather than by an atheroma of the intima; and at one spot was found an aneurysm the size of a cherry. The coronary orifices were blocked. Yet there was no history of angina.

The conclusion then to be drawn from these further clinical and pathological records is, that if angina arises during an ordinary pericarditis it is due to an extension of it around the collar of the great vessel; that if it arises on embolism or swift thrombosis of a coronary vessel it must be of a main or large trunk, and the pain due to tension of the investment of the

[1] Weber, Parkes, *Path. Trans.*, 1896.

heart either by pericarditic products or by the engorgement of the peripheral area beyond the plug.

Death by Inhibition.—But how then, on this aortic hypothesis, comes angina pectoris to be so fatal? In this vessel are not dilatation and disease common enough, and, if part of the machinery of death, nevertheless not lethal in this dramatic sense; how indeed can aortic disease, without rupture of the vessel, prove thus suddenly fatal? This difficulty however we have to meet in any current interpretation of angina. How is it that an attack of pain at length kills suddenly, and by a heart-stroke, although time after time physicians have watched the invasion to the very door of death without a totter of the heart; or with no more perturbation of it than is seen in many a transitory disorder which, apart from the signals of angina, would not give us even momentary alarm? Is it not still more incomprehensible that often, while the patient is in so hard a grip that he cannot start back even from death's door, his pulse may be going on its way with indifference, and not be moved even with a secondary and sympathetic affection? Thus I was led to surmise that in angina pectoris the fatal shock must be something secondary and quasi-accidental, and then I thought of an inhibition of the heart by reflex action, by a kind of "shock."

It is thus that the swift bolt of angina, often as silent as swift, striking a man down in his path, has possessed all who have known it with a kind of awe. The physician perceives at once that this swift passage is by the heart; hence Parry's name of *Syncope anginosa*, and that of *Sternodynia syncopalis* (Sluis, 1802). That this death is due to the vagus, stung by the anginal pain and acting often upon a decrescent heart, is the part of my hypothesis of angina (1894) which already has found some if not general acceptance.[1] In order to understand how death

[1] There is a legend, apparently originating from von Bezold, that Rufus of Ephesus and Galen knew of the influence of the vagus upon the heart. Rufus (after Erasistratus) certainly described the nerves springing from the brain, but of this special effect of the vagus I can find nothing. To say there is no such statement in Galen is more than I dare assert; Galen is a copious person; but I do not recollect any such passage. Before the brothers Weber (1846), Willis and Lower (end of seventeenth century) showed that to cut the vagus

happens in angina pectoris—for at first sight death is not, I repeat, a likely issue of aortitis—let us consider, briefly as it must be, some relevant points in the mechanism of cardioarterial sympathies and concerts. In the first place we shall *distinguish between the vago-sympathetic and the vasomotor mechanisms*, the centres and the branches of which form distinct and partially independent systems. The superficial and the deep cardiac plexuses lie, as to the former between the pulmonary artery and the aorta, and as to the latter and much larger portion, on the posterior part of the root of the aorta; these portions are however so intimately connected that for present purposes they may be taken as one.

The vago-sympathetic group consists, as the name implies, of contributions from sympathetic and vagal sources; and from this subcentre fibres are distributed to all parts of the heart and contiguous vessels. It contains afferent fibres which pass to the spinal cord by the posterior roots and thus attain relations with consciousness. There are probably afferents from the whole of the aorta, and from other arteries—reflex or automatic, local or general,—but little is known of them. The sympathetic contains vasoconstrictor fibres. Moreover, as will be illustrated presently by some observations on the pleura, there is a connecting link between the cardiac and pulmonary plexuses, so that irritation in the one is felt more or less vividly in the other. The centre for these plexuses in the bulb is distinct from that of the vasomotor system, although a heightened activity in either may be felt by the other. Furthermore, as we shall see presently, these centres are in close concert with the respiratory centre in the bulb, and have with it harmonious periods of activity and relaxation (see also fifth nerve, p. 297). There are of course vasomotor centres in the thoracic cord also, with connecting fibres to the sympathetic.

The vasomotor connections of the heart, for the later elucidation of which we are indebted to Professor Langley's researches, are supplied from the sympathetic system. At present we may confine ourselves to its splanchnic division, and to its

quickened the heart; and ten years before the Webers Volkman made experiments on the vagus with an electric stimulus, and included it in his compression experiments.

regulation in respect of the heart by the depressor nerve whose action, passing first in an afferent direction, then causes a dilatation of the splanchnic reservoirs, whereby at need peripheral resistance is lowered, and the heart is relieved of stress. The vagus and depressor nerves are no doubt both in incessant touch with the heart and the aortic tambour, as the hand of a good horseman is in continuous touch with his horse's mouth. For inhibition, if we think of it, is not an occasional interference with vital function but a primary condition of it; without it energy would be dissipated as soon as conveyed; there would be no storage of energy. Indeed inhibition is not unknown in the sphere of chemistry, as with orcein and resorcin. Biological inhibition may depend likewise on some chemical phase. Sir David Ferrier has spoken of the depressor, "the afferent nerve of the heart *par excellence*, as in reality the sensory nerve of the aorta."[1] We know how Ludwig and Cyon demonstrated the two roots of the depressor, one from the vagus in the neck, the other from the n. laryngeus superior, which goes to the cardiac plexus. To irritate the central end causes a fall of pressure and retards the pulse; but if the vagus be cut only the former effect is seen. The depressor is thus partly vagal, partly vasomotor, chiefly splanchnic, but with some peripheral distribution. It is a safety valve for cardiac distension. Here also I must refer again to Köster and Tschermak's experiment of isolating the aorta and demonstrating that distension of its first portion is followed by a fall of blood pressure.[2] Schumacher,[3] who was working independently of Köster, showed also that it pertained to the first portion of the aorta. But we must remember not to apply too readily to man results obtained upon animals; there are grounds for analogy, or even homology, but probably not for identity. Professor Sherrington also[4] speaks of the aorta, as for many years I have done, as the receptive field for the depressor whose terminations in the wall of the vessel have been demonstrated; and its most adequate stimulation is a distension of this wall, whereupon by relaxation

[1] See also Köster, "Ursprung d. N. depressor" *Neurol. Centralblatt*, 1901, p. 1032.

[2] Köster and Tschermak, *Pflügers Arch.* Bd. xciii., 1902.

[3] Schumacher, *Sitzungsber. d. Wiener. Akad. d. Wiss.* Bd. iii. Abt. 3, 1902.

[4] Sherrington, C. S., *Brit. Med. Assoc.*, 1909.

of vascular tone arterial pressure falls. But such stimulation does not cause pain—at any rate, not directly.

If the pain of angina excite both the vagus and the vasoconstrictors, it appears—*e.g.* under adrenalin (Schäfer, *loc. cit.*)—that the vasoconstrictive action predominates; although the vagus may still be in action. When it is severed the blood pressure rises enormously. I do not know that it is always true to say that this double effect of the pain makes for more embarrassment of the heart. An intraventricular rise of pressure may call forth a stronger cardiac response, such as not only to resist the nursing vagus but also by rise of tension to stimulate the depressor. These considerations apply only to immediate effects. In the long run, section of the depressor (in dogs), by increasing the call on the heart, brings about a genuine hypertrophy both of heart and aorta [1] (media and intima); and in hyperpiesis the vagus seems to get used to habitual excesses of blood pressure. I cannot agree with Bittorf that this ease is due to lesion of the vagus terminals. But all depends upon the capacity of the particular myocardium, if old or young, sound or unsound. Bafflingly complex are all these coefficients. If the vagus is intact the pulse is slowed, as we may note occasionally during attacks of angina (Vol. II. p. 332); but the prolonged fall of pressure is not due to this—a longer diastole means at first a larger contraction volume—but to dilatation in the splanchnic and probably some other areas also.[2] So if, momentarily, this and the vagus diastole may mean plus output, this soon ceases as less venous blood is returned. If the splanchnic nerves are divided, the corresponding area fills with a large bulk of blood, which is thus removed from the active circulation. A further and perilous relaxation of the splanchnic system in shock and collapse is, up to a certain point, too well known to detain us in this place. Again, if the restraining influence of the cardio-inhibitory nerves is removed by division on both sides the heart begins to gallop. But, although we cannot but argue severally about these divisions of the nervous endowments of the heart, yet in life their connections are so complex and intimate

[1] Bruns u. Genner, "Einfluss d. Depressors auf die Herzarbeit," *Deutsche med. Wochenschr.*, No. 37, 1910.

[2] See Bayliss, *Journ. of Physiol.* vol. xiv., 1893.

that to alter the balance in any one of them operates as a compound effect. This integration is true especially of the augmentor nerves which, broadly speaking, on their excitement serve to increase the output of the heart even, within limits, against a rise of peripheral resistance. The functions of the vasomotor and vagal divisions are however more independent than that of the augmentor; and, under experimental observation, the normal inhibition of the heart by the vagus, mixed nerve as it is, may be made plain. For instance, during experimental rises of arterial pressure which, forasmuch as its own arteries are not, or are but little, subject to waves of constriction, are felt at once in the medullary centre, the curb upon the heart becomes instantly perceptible; its work is diminished and, *caeteris paribus*, the pressure falls to its former level. So dependent is the circulation upon its nervous adjustments that Balint,[1] in some experiments in dogs and cats, found that to destroy the aortic cusps produced no drop of function, and gradually the heart became hypertrophied. So again, the fatty heart produced in dogs by phosphorus does not make at once for functional failure; yet in dogs, even with a sound myocardium, to cut one vagus, and still more to cut both, ends promptly in heart failure.

It seemed necessary thus at some length to reconsider the sphere of action of the vagus in order that my hypothesis of its potency in angina might be better understood. The function of the vagi, conservative as within limits it is, may nevertheless, as Goltz, starting from the original observations of the brothers Weber, in 1845, proved on cold-blooded animals, have a mortal issue; especially when the heart is old or enfeebled. A like effect was then obtained by experiment in warm-blooded animals on *stronger irritation, not of the substance of the viscera but of their investments*. When the vagus is cut this effect is prevented. However, by virtue perhaps of its mixed character,[2] the vagus thus vexed does not usually stop the heart, not even if the irritation be continued; the vigorous heart escapes from its governor, and in a strait refuses to spare itself at the cost

[1] Balint, *Deutsche med. Wochenschr.*, Jan. 6 and 13, 1898 (quoted by Dr. J. H. Sequeira).

[2] Some experiments suggest that the vagus may contain some accelerator fibres, the action of which is usually masked (see Laidlaw and Symons, *Journ. of Physiol.* vol. xli., 1910).

of the life of the system. Indeed ordinarily, if the stimulus cease, the inhibition of the heart beat is followed by a rebound. Hence the extrasystoles not infrequent in angina. Cushny and Edmunds [1] have shown that the vagus effects its purpose by lowering cardiac conductivity (heart block); and in a more recent study of the subject, Hering of Prague [2] confirms this opinion, with further detail, some of which concerns us here. But, as Dr. W. T. Ritchie says,[3] the lowered conduction gradually ceases, even if the stimulus be continued. I have sometimes speculated on the effect in the heart of sudden change of motion into heat, which might aid the accelerators in this "escape." That in pyrexia the heart is accelerated we know, but here other causes of acceleration come in. James and Williams [4] note that in the electrocardiogram a heart under vagus preponderance shows only a prolonged diastole; and it is in the later part of diastole that the vagus tells most effectually. It is in this phase of angina, not "in systole," that death takes place. A fall of pressure, as by mitral regurgitation, or by means of a drug, *caeteris paribus*, disarms the vagus, and averts death by inhibition; and this in angina we commonly see (p. 443), and even bring about, as by the nitrites.

I have said that during an attack of angina, sometimes plainly sometimes less distinctly, the pulse may alter to the touch; it may hesitate or fluctuate, usually by retardations suggestive of effort or check (p. 332). To Dr. Mackenzie's remarks on pulsus alternans in angina I have made some allusion (p. 334); it is important to distinguish this falter from the controlling influence which I am discussing. And both these conditions are to be distinguished from the retardation of heart block. Dehio, twenty years ago, and Kronecker [5] also, discussed this last kind of retardation as a loss of irritability or conductivity; Gaskell,[6] Roy, and others

[1] Cushny and Edmunds, *Amer. Journ. Med. Sci.*, 1907.

[2] Hering, H. E., *Münch. med. Wochenschr.*, 1910, No. 37, and *Zeitschr. f. exp. Path. u. Ther.* vii. pp. 363, 1909.

[3] Ritchie, W. T., *Quart. Journ. Med.*, 1912, vol. vi. p. 47.

[4] James and Williams, *Amer. Journ. Med. Sci.*, Nov. 1910.

[5] Kronecker, *Arch. f. Anat. u. Physiol.*, 1883, p. 263.

[6] Gaskell, *Journ. of Physiol.* iii., 57, 1880; and *Phil. Trans.*, 1882, p. 1017 ("that the excitability of the ventricular muscle is at the time not absolutely the same throughout; certain portions respond only to every second impulse.") See also for recent research Mines, *Journ. of Physiol.* vol. xlvi., 1913.

have discussed pulsus alternans as an expression of differential or fractional muscular states, and of remainder blood in dilated, and not dilated, cavities respectively. Then Hering, verifying Gaskell's explanation, emphasised the distinction of pulsus alternans from extrasystole which might simulate it.[1] But Dehio was, I think, the first to use atropine as a means of analysis, so as to distinguish vagus from myocardial lag; this valuable test often enables us to discern in a particular case how much of the retardation may be myocardial or Purkinjean, and how much, if any, due to vagus, and therefore transitory. Under atropine the dissociation of auricle and ventricle may be ended, or at least modified; the auricular rate may rise while the ventricular remains unchanged; and conversely, as Thayer and Peabody have demonstrated,[2] to stimulate the vagus in heart block—as by pressure in the neck—may throw out ventricular beats and slow the auricular even without notable prolongation of the a-c interval. But if the heart block be wholly myocardial, if, that is, the dissociation be more or less structural, atropine may make no difference. We shall see under the head of Treatment why I am labouring these points, and how it is that this action of atropine is of great practical importance. But Spiess and Magnus-Alsleben [3] of Basel, who produced pulsus alternans in rabbits by "glycoxylsäure," concluded that, generally speaking, pulsus alternans consists not in partial asystoles but in a universal hyposystole. And they found also that, under these circumstances, the vagus might even prevent a pulsus alternans; for on section of the a-v bridge, so as to isolate the ventricle, the pulsus alternans ceased at once ("trat prompt auf"). As Thayer and Peabody say, at times during an attack of angina one may feel alternating waves of vagus influence; waves which may or may not sum with other factors to modify the phrasing, auricular or ventricular. And I have seen, on accelerating the rate by atropin, that with this acceleration pulsus alternans may become perceptible, although apparently not continuously present. Pulsus alternans then is no simple state of myocardial affection, but is a complex of variables, and not necessarily persistent.[4] So in

[1] Hering, *Präger med. Wochenschr.*, 1902.
[2] Thayer and Peabody, *Arch. int. méd.*, March 1911, vol. vii.
[3] Spiess and Magnus-Alsleben, *Zeitschr. f. exp. Med.* Bd. ix. Heft 2.
[4] Wenkebach (*Unregelmässige Herztätigkeit*, 1914) has submitted pulsus

angina, if there be any degree of the Stokes-Adams condition, what I have called a phrasing of the pulse may be observed; moments of summation of stimuli, a full consentaneous contraction with dissipation of waste, and then a falling away of this integration. But even when dissociation is present the a-c interval may still be normal (Wenkebach, Gossage, and others). Once more: pulsus alternans may occur with little or no default of the heart in cases of high aortic pressure. At moderate pressures the elastic vessel in easy diastole and systole takes up and neutralises variations of output; but when the wall is very taut this play approaches to zero, and thus unyielding does not take up these variations; now every output, plus or minus, is reported in the pulse, and such an alternation is not uncommon, and need not, I think, signify a failing heart. Of course, during brachial compression, in obedience to psychical causes or to respiratory tides, radial beats may come through unequally.

Hay and Moore[1] also have demonstrated, by Mackenzie's method, how salient may become the influence of the vagus in the Stokes-Adams condition; and there is other evidence to prove that myocardial degeneration does not impair the control of the vagus, that on the contrary such a heart is stopped by it more readily.[2] Dr. Mackenzie says that "if any one function of the heart is depressed the vagus is prone to seize upon it, and to increase the depression"; and again "depressed cardiac functions are more susceptible to vagus stimulation." Dr. Gaskell also showed, what is of great importance in this study, that after vagus stimulation the restoration of cardiac function is gradual, the beats increasing in strength slowly as the anabolic and fortifying effects are realised. This, as he says, is gradual function; but if the vagus be hit by what I may call a snap-shot the heart is checked without recompense, the sudden sharp stimulus to the vagus having more of the depressor effect and less of the anabolic and restorative effect than consists with more gradual interference. And herein may lie some explanation of the reflex vulnerability of old or diseased hearts,—that in them the anabolic restoration is still slower of attainment; nutrition

alternans to clinical analysis, and considers that Mackenzie's view of it as a foreboding of death needs qualification (see case, p. 265).

[1] Hay and Moore, *Lancet*, Nov. 10, 1906.

[2] See Dr. Anderson's Cat Experiments, p. 475.

is sluggish, and osmosis and surface tensions are in the older tissues less vivid. Dr. Lewis[1] agrees that in prolonged a-c interval (partial heart block) the vagus effect may be instantaneous: the next ventricular contraction is suspended, and no response may be obtained till several auricular systoles have passed. We may note by the way that the a-v bundle is said to be nourished chiefly by a particular branch of the right coronary artery, a point to be observed in cases of death with partial coronary obliteration.

Thus, under many known and some unknown conditions the heart, as in the familiar Goltz experiment of tapping a frog's abdomen, fails thus to escape. *It is then in its first impression that the vagus influence is most critical;* as its influence is protracted counter influences gain ground; thus, if the heart is sound enough to hold on, it gets driven forward.[2] It is said that as the heart is escaping from vagus inhibition the curve is, or often is, anacrotic;[3] a minus cardiac impulse being, in respect of this phenomenon, equivalent to plus aortic resistance. In the frog experiment the vagus influence is sudden and maximal, conditions which even for a healthy heart may become irresistible (*vide* Reich, p. 483). Still, the healthy heart of young subjects, as in the angina of syphilitic or rheumatic aortitis, manages nearly always to escape in time—its readjustments are both elastic and ready; with the deteriorated or even with the merely old heart it is not so. As the active youth slips his foot and promptly saves himself, but the stiffer joints and unready muscles of the elderly man fail to respond in time and he falls before he can save himself, so it is with the heart imperceptibly altered by age, or enfeebled by coronary or other disease; it cannot save itself, it topples, regains a step perchance, loses it again, and drops. Thus occasionally in a sound patient by extreme agony, by an agony such, let us say, as the passage of a calculus, a blow on the testicles, or a sudden and intense emotion, the heart may be inhibited to fatal arrest; as

[1] Lewis, T., *Quart. Journ. of Med.*, Jan. 1911.

[2] Lewis, T., *Practitioner*, Feb. 1907.

[3] Von Frey has shown that an anacrotic curve may be noted as the heart is escaping from vagus inhibition, and that, as Roy and Adami and Lewis had proved, such a curve may be one of only relatively excessive peripheral resistance; *i.e.* of heart weakness.

again it may be by a specific effect of chloroform upon the vagus. In the vagal nucleus, says Sir David Ferrier, a pin-prick will arrest the heart. It is said indeed that firm pressure upon the eyeballs will inhibit the heart, even to syncope, enfeebling, retarding, or even arresting the radial pulse ; and that in animals (dogs) the heart may thus be stopped in diastole. It is more than probable that, *in the degree of deterioration which an aged or diseased heart may have undergone, its chances of escape from an irritated vagus are proportionately diminished.* Besides, we shall see presently that *in angina the vagi become hyperæsthetic* (Wenkebach).

Dr. Hugh Anderson, to whom I desire again to express my thanks, showed to me a very instructive experiment bearing on this point. From a cat he removed the third—accelerator—cervical ganglion ; so that the heart was delivered over to contrary influences, as of the vagus. Now it appears that if, taken in the hands, this animal be waved up and down, the vagus is stimulated to action. In a young cat thus operated upon and, on recovery from the operation, waved up and down, the effect of the vagus may not be conspicuous ; the heart may lag a little but it is not much affected, and never arrested. But if the cat be an old one—one however in which on autopsy cardiac degeneration may not be manifest, its life is in perpetual danger; to wave it now reduces the ordinary cat pulse of 120 perhaps to 40. And not only may the waving kill it, but it is in peril of death from ordinary contingencies. So liable indeed is the animal to die suddenly that further observations often come thus to a premature end. For, while the vagus resents its stimulus promptly, reflects it as instantly, and on the removal of the stimulus as promptly releases the check, with the augmentor and accelerative influences it is otherwise. These agencies come into action gradually ; they have a long period of latency, a slow approach to the maximum, and a long persistence after removal of the stimulus (Gaskell and others). Thus experiment verifies what clinical experience had abundantly illustrated, that under inhibitory influences an old heart, even though to the eye of the pathologist it may not betray disease, may yet stop while a young one escapes.

But this is not all ; in angina the vagus is morbidly sensitive,

as one may say—worried. We may be surprised in angina to find this nursing nerve become so ascendant, and so menacing, seeing that in other diseased states—in uræmia for instance—it may retard the heart but does not arrest it. Some light is thrown upon this point by a letter of Professor Wenkebach to myself, in which he writes that in angina he has found the vagus extraordinarily sensitive.[1] In a few cases, during tranquil intervals, he has very cautiously tested it by weak pressure on one side only, and has found the effect to be prompt and excessive. As an afferent, it seems in this disease to be baited to a degree of dangerous activity. Indeed he thinks that the heart of an anginous patient might be stopped by a tight neckband. In such states close observation of the vagotonic motions of the pupils of the eye might assist us, especially during deeper respirations. Sir Edward Schäfer[2] also points out this further peril of sudden death. The inhibition may be deferred, but in two or three minutes "suddenly show itself and prevail over the accelerators," when the auricles may cease to beat, and the ventricles contract with a slower and somewhat irregular rhythm; points which remind us of no infrequent experience in angina pectoris. To meet this abatement the accelerators and augmentors, acting upon both auricles and ventricles, renew the rate and force of the beats, and output is increased, even against some rise of blood-pressure. This effect, he adds, is "precisely similar to electrical excitation of sympathetic branches proceeding to the cardiac plexuses." I may repeat that, while the accelerans reinforces both systole and diastole, the vagus slows diastole only; hence its occasional mistakes. In clinical practice we perceive darkly how complex are the conditions at work, excitatory and inhibitory influences blending variously and inconstantly as preponderances, quotients, or resultants; and subject meanwhile to preventions of access not only by antagonisms but also by temporary or permanent changes in tissue conductivity.[3]

[1] I find the same observation is made in certain cases of heart disease by Robinson and Draper, *Journ. Exp. Med.*, Jan. 1, 1812, vol. xv. 1. As these pages are passing through the press Professor Wenkebach has published these clinical tests, with graphic illustrations, in his new work on the whole subject of irregular cardiac action, Engelmann, Leipzig u. Berlin, 1914. He accepts my hypothesis that sudden death in angina is by vagus reflex.

[2] Schäfer, Sir E. A., "Functions of Suprarenal Capsules," *Brit. Med. Journ.*, June 6, 1908.

[3] See Sherrington, *Proc. Roy. Soc.*, Dec. 1908.

To turn now to the vasomotor or *depressor division*; here we find that the relief to the heart is by a direct relaxation of pressures on dilatation of the vessels of the abdominal viscera, which may become heavily engorged; accordingly here too the heart's work falls in output as well as in diastole. Within the normal play of our reciprocating engine, as Professor Sherrington has so admirably demonstrated in the skeletal muscles, inhibition diminishes tonus in antagonists; if then we regard the vasoconstrictors as antagonists of the ventricular muscle, we may anticipate the explanation of reciprocal working on vasomotor governance to be analogous to that in the voluntary muscles. "Two antagonists may be in action together, but their activity decreases and increases in reciprocal proportions; hence the smoothness and accuracy of trained movements" (Sherrington). Hirsch and Städler think the depressor, which by twigs from the jugular ganglion is a sensory nerve of the aorta, does not exercise this continuous reflex, but regulates rhythmical efflux on systolic call only. There are of course other depressor nerves also. How readily a lysis of the circulation may come about through the depressor I need not stay to explain; indeed it is the less necessary to do so as in angina pectoris I do not suppose this to be the usual mode of dissolution; in this disease death is ordinarily by the alternative peril of vagus arrest. Von Dusch, it is true, spoke of death in angina by paralytic dilatation of the heart, but quite conjecturally.

François Frank, in Marey's Laboratory in 1876,[1] showed how irritating vapours, when applied to the nasal and laryngeal mucous membranes, would retard the heart through the vagus; observations which were repeated and enlarged by Brodie and Russell.[2] These observers proved that almost any afferent nerve of the body, under appropriate stimulus, can retard or arrest the heart. They showed then, what is now well known, that the stimulus of a rise in arterial pressure will, *caeteris paribus*, retard the pulse. And we know also that cortical irritation may have similar effects. Dr. Jex Blake has repeated these observations.[3]

In the Laboratory of Experimental Therapeutics in the

[1] Frank, François, *Acad. des Sciences*, 4 déc., 1876.
[2] Brodie and Russell, *Journ. of Physiol.*, 1889.
[3] Jex Blake, *Brit. Med. Journ.*, Jan. 21, 1911.

University of Chicago, Drs. Capps and Lewis [1] have illustrated these processes in a very interesting way. In my early Leeds essays on empyema, I was led by experience to deprecate the use of intrapleural antiseptic injections, because under such interferences, and apparently in consequence of them, syncope or even death had happened.[2] In some of my own cases under such washing alarming faintness had occurred. Many records were published later of these perilous events on washing out the pleura or otherwise interfering with this cavity, especially near the root of the lung; *e.g.* by Cayley, Goodhart, Raymond, Vallin (who repeated Capps' and Lewis' experiments), and others.[3] As such injections often seem desirable, these warnings were received by physicians with some reluctance; and the deaths recorded in certain cases were attributed, not to this operation, but to thrombosis or some other accident. As years went on however more and more cases were added to the lethal list, and the practice of injection began to wane; but, although Dr. A. E. Russell had in some measure anticipated these results, and had thus explained a case of sudden death on an exploratory puncture of the chest by cardiac reflex,[3] I think that the Chicago research was the first definite experimental proof of the chain of causes. Russell did not find the pleura so sensitive as did Capps and Lewis, but these observers noted its response especially when the pleura was inflamed. Dr. Johnston [4] has since demonstrated the fine nerve ramifications, which he regards as the paths of pleuritic pain, in the suprapleural connective tissue investment. But with the lessons hence to be learned in the practice of thoracentesis, impressive as they are, I am not immediately concerned; my purpose is to direct attention to the cardio-inhibitory effect of certain peripheral irritations, especially of the susceptible thoracic areas, and in them of investing tunics.

[1] Capps and Lewis, *Amer. Journ. of Med. Sciences*, Dec. 1907, and *Arch. of Int. Med.*, Dec. 1911. See also Brodie and Russell, *Journ. of Physiol.*, 1900; and Cadman, *Journ. of Physiol.*, 1900.

[2] See Editorial, *Lancet*, Sept. 28, 1912, but I had published this warning, based on experience, long before "Dr. George Carpenter in 1893."

[3] *Vide* A. E. Russell, St. Thos. Rpt., 1899, and *Clin. Journ.*, Sept. 8, 1909. But in 1864 Roger had recorded an eclamptic attack which occurred during operation on a case of empyema (p. 480).

[4] Johnston, *Brit. Med. Journ.*, September 11, 1909.

Capps and Lewis began by quoting the experience, now perhaps generally accepted, that in surgical operations about the pleura "there still remains a considerable number of cases of sudden death, in which no anatomical lesion (such as embolism) can be found post-mortem to explain the collapse." Of the connection between the cardiac and pulmonary plexus I have already spoken: now Capps and Lewis tested the cardiac response upon this route, and demonstrated that, although on irritation of the normal pleura none, or but little, of such an effect may be perceptible, yet in inflammation of the pleura a potent effect is manifest. The effect is usually of the cardio-inhibitory kind; the heart is slowed, and the pulse tracings make wide excursions between systolic and diastolic pressures; the respirations also are usually slowed, and for the moment may be arrested; a point in angina to which, under the head of Symptoms, I have referred (p. 313). By atropine, or by division of the vagi, they could prevent these effects. But occasionally the influence stole rather along the splanchnic vasomotor division, when the pulse curves betrayed a rapid decline of both systolic and diastolic pressures; the respirations became shallow and often rapid, and the abdominal viscera engorged. This even more dangerous effect they could prevent by intravenous injection of adrenalin. By carefully watching operations for empyema in man, and by noting especially the effects of scratching the inflamed parietal pleura, sometimes the one and sometimes the other of these effects was observed; sometimes the pulse became slow, even to intermittency; sometimes pressures and output fell together, and the pulse dwindled to nothing. The afferents, as we have just seen, are distributed, as Lennander demonstrated for the peritoneum, not to the serous surface, but to the areolar tissue immediately beneath it.

In a few instances a vasoconstrictor action, occasionally to a well-marked and persistent degree, was observed; but this was quite exceptional; such constrictive rises of pressure, if recorded at all, were transient. Why in the many cases the vasomotor result should be relaxation, but in a few constriction, is obscure; but it is notable that the constrictive effect was more frequently observed in the cardio-inhibitory cases. In this respect we may recall to mind that in most cases of migraine, or in suc-

cessive stages of a single case, such alternations of vaso-constriction and dilatation are likewise to be observed. As we have seen concerning the peripheral pleural surfaces, the inhibitory effects were obtained vividly only when these tracts were inflamed, and the vagus annoyed; but as the root of the lung was approached the cardio-inhibitory effect became so much the more that it would manifest itself promptly even without any inflammatory factor: there the stimulus was approaching the pulmonary plexus, and the pulmonary is connected with the cardiac plexus into which the vagus enters. Brief cardio-inhibitory effects, single prolonged heart strokes such as I have noted occasionally in the pulse of angina (p. 332), could be called forth almost at will, by stretching the root of the lung, or the pericardium; or by scratching the thoracic portion of the vagus. Section of the vagi in the neck, or an injection of atropine, would stop the cardiac effects, but did not exclude a splanchnic dilatation; so that a valuable differential test of the nature of the cardiac effect was thus obtained. In 10 per cent of operations on the pleura and lung, Körte and Lenhartz noted some degree of cardiac inhibition—a transient retardation of pulse and respiration; and Brodie and Russell demonstrated that irritation of the pulmonary efferent fibres of the vagus at the root of the lung arrested ventricular contraction and respiration with fall of blood pressure.[1] Indeed in 1864 Roger had made observations of the same kind on an eclamptic attack which occurred during an operation on a case of empyema.

Startling effects then may follow even a simple pleuritic operation. Brunque and Zesas[2] collected reports of 21 deaths on incision, on lavage, or even on simple puncture (a case of embolic hemiplegia was omitted). Gunshot and other accidents causing costal osteitis, or tapping for a cancerous effusion, have had syncopic, eclamptic, or mortal effect. How important a bearing these observations have on my interpretation of death in angina pectoris, the reader probably already apprehends.

On hypothetical grounds, in more than one previous paper on the crisis of life in angina pectoris, I have recommended artificial

[1] Brodie and Russell, *Journ. of Physiol.* vol. xxvi. p. 92.
[2] Quoted in an editorial note, *Semaine méd.*, 27 nov., 1912.

respiration; and Brodie and Russell have shown that this operation tends to quieten reflexes from irritated viscera. During Capps' and Lewis' experiments the thorax was laid open.

The next illustration of this kind of inhibition which I will produce, and many more are at our service, is taken from the Address in Surgery at the Meeting of the British Medical Association in 1904. Sir William MacEwen then referred to certain observations which he had made upon a sympathy between the pudic and respiratory tracts, a response being obtained instantaneously. When the pudic periphery of a patient, under anæsthesia, is irritated, a prolonged, almost tetanic, respiratory gasp may be produced; and on each single repetition of the irritation a stridulous inspiratory effort follows. In the majority of cases the heart yields, if at all, so slightly to this vagus reflex, that to the finger on the pulse the reduction is scarcely perceptible; in some instances however the cardiac sympathy is more ready; coincidently with the stimulus the pulse weakens. In a few persons this effect is still more remarkable and ominous; evidently by the faltering of the pulse and the livid pallor of the face it may become perilous. And here again these phenomena, if not very notable while the tissues are normal, are rapidly enhanced under an inflammation; as may be observed in the parallel case of an inflamed joint, irritation of which under anæsthesia may set up spasm of the corresponding group of muscles and rigidity of the limb.

Sir William Gowers, in the "Vaso-vagal Essay" to which I have often referred, says, "In man, I think, a cause of adequate pain has not been known to produce syncope if the patient were under the influence of an anæsthetic"; and he surmises therefore that the afferent impression may be not on the vagus centre, or not there only, but rather on the sensory regions of the cortex, and thence focussed down on the cardiac centre in the medulla, as in cases of sudden intense emotion. For my present argument this question is not urgent; but I may remark that, whatever the mechanism, no such protective effect of the anæsthetic is notable in operations on empyema, on the brachial plexus, or (as regards those which I have quoted) on the pudic area; nor in my experience is complete protection notable in experiments upon animals, even when under the deepest anæsthesia. Quite

recently Crile and Cushing, under the awkward title of "Anoci-(Innoci- ?)-association," have done much to elucidate this subject. Morphin and atropin seem to afford substantial protection.

But, leaving this point undecided, I would reiterate the inference which seems to be enforced by all these observations; namely, that when the peripheral tissues which accept the irritations are inflamed, such reflexes are much intensified. Now it is in such an irritable state of the aorta, and especially of the supra-sigmoid portion of it, whether in acute aortitis, or a subacute extension of chronic syphilitic or of atheromatous disease, that we may suppose this peripheral area, and probably the vagus, to be in an exalted state of susceptibility, highly obnoxious to impressions, and highly conductive of afferent currents, and that the corresponding centre has become abnormally irritable. As Professor Barker has said (B.M. Assoc. 1909), and as in Dr. Anderson's cats, "syncope is very likely to follow slight violence, even without operation, in old or decrepit people," the enfeebled or old heart being more susceptible to inhibition, and less able to resist it. As with voluntary muscles (Sherrington), fatigue and certain drugs—*e.g.* chloroform—are conditions favourable to inhibition. Moreover, according to circumstances, certain stimuli (*e.g.* toxic) may promote either excitation or inhibition, whereby unforeseen reactions may ensue. Besides the surgeons mentioned, Mummery[1] has thrown light upon the perils, in the course of an operation, of dragging upon or wounding certain nerves; perils which may be even mortal. Now from our present point of view it is important to note that the brachial plexus is a way peculiarly open to this dangerous reflex; even to sever its strands, as for example in an operation for cancer, may cause sudden pallor, and failing of the pulse even to disappearance at the wrist. Usually the effect is but momentary; the colour soon returns to the lips, and the pulse improves: but the issue may be less happy; the pulse may fail to get back, and the arterial pressure may drop lower and lower to death, a death which may be wrongly attributed to the anæsthetic. Mr. Mummery published a chart of pressures during an operation in which traction on the brachial plexus caused a fall in pressure from 150

[1] Mummery, *Lancet*, March 25, 1905.

to 50; the pressure rising again quickly to 100. To crush the testicle or the paw of the dog, although under an anæsthetic, thus abases the blood pressure; at the first moment there is a transient rise, then the fall follows. As one step farther, Reich [1] has given a full study of operative interference with the vagus itself. He showed that clean section only paralyses it; but crushing or dragging, or even the touch of forceps or bistoury, re-excites it, so that in critical cases he recommends preliminary resection of the nerve of the side. From his summary, in six propositions, only the fifth concerns us here: that crushing of the vagus is followed by inhibition of cardiac and respiratory action, which in man may be mortal. For example, Dr. Falkner Hill of Manchester sent me notes of two events of this kind during an operation for extirpation of a malignant thyroid. The anæsthetic was 1½ per cent chloroform, with ether and oxygen. In spite of due support of the jaw, it was dragged down, when, the patient's condition having till then been quite normal, the heart and respiration stopped. Restorative measures were successful, and a new start was made without any chloroform admixture; but, on another unavoidable traction, the same events recurred, with a mortal issue. From my therapeutical experience in angina, I suspect that atropin would prevent these accidents; but, for operation purposes, to block the afferent path by morphia seems generally to be efficacious. In comparative experiments on animals, Reich found the human heart more susceptible to excitation of the vagus than the animal; it proved to be impossible thus to cause the death of an animal.

Now as chloroform is in common use for the relief of protracted assaults of angina, it is a serious matter for us to know if this anæsthetic, or chloral, may be a treacherous ally. I am by no means sure that, in so nice a balance of forces, chloroform is a safe remedy; in the abstract it is not; in my experience morphin is no less efficacious, and, moreover, avoids irritation of the nasal afferent surfaces. In a paper read to the Royal Society (*Proc. R. Soc.* 1906) Dr. Embley of Melbourne stated that, in the dog, chloride of ethyl stimulates the vagus, and, as the drug becomes more concentrated, the heart halts under vagus inhibition; yet not fatally. As in the animals observed the hearts were

[1] Reich, "Beiträge z. klin. Chirurgie," quoted *The Hospital*, Jan. 7, 1908.

healthy, we were prepared to hear that in each case the organ escaped from the influence. Now in the dog chloroform "produced an equal effect in one quarter the concentration in air"; not only so, but inhibition was succeeded by paralysis of the heart, and arterial pressure fell and fell until respiration ceased. Dr. Embley stated that he had witnessed cardiac syncope under chloroform in two cases in man; though happily in both recovery set in, with sudden return of the pulse in fair volume, and of the living colour to the face. A sound heart, he added, can generally free itself from vagus arrest, but a feeble heart may fail to catch hold again on life. Morat and Doyen, Brodie and Russell, testify also to these effects; and the comprehensive study of this subject for many years past by Dr. Waller is too well known to every reader for me to do more than allude to it. In this paragraph I am alluding only to early and sudden deaths, often under light chloroform anæsthesia; deaths indirectly due to chloroform at later stages, deaths due to intoxication of the cardiac muscle, or to "acidosis," do not belong to my subject.

Now let us apply these results to disease and to angina pectoris. Perhaps all the arteries have their degrees of sensibility, but the suprasigmoid portion of the aorta, and probably other parts of it likewise, especially perhaps the cœliac portion of the abdominal aorta are normally endowed with a peculiar afferent sensibility to blood pressures. In such a part, let us suppose a sore spot, an acute inflammation, or a subacute extension of a more sluggish process creeping over it or into it. As we have seen, such a condition of receptive tissue is extraordinarily quick to promote and to multiply reflex phenomena, and to accumulate stresses in its controlling centre. If such a reflex be focussed upon a sound heart, in a youth with rheumatic aortitis, let us say, or in a man in the prime of life with influenzal or syphilitic aortitis, the heart, although touched unpleasantly or even critically, will generally escape. As we have learned concerning the cardio-reflex effects obtainable in even healthy animals by irritating or dragging upon the central end of a divided nerve, such as the sciatic, so the stories are many of sudden death in young and healthy adults from some swift and poignant pain. And if we suppose the heart to have become enfeebled by

decay, due perhaps to a failure or perversion of blood supply, or to age, even then it may escape once and again, but sooner or later will probably be overthrown. In such observations and experiments as I have quoted we see that the critical moment for the heart in angina, as in light chloroform anæsthesia, may lie in a first touch; let the organ have time to call up its reserves and its compensatory mechanisms, and it may be able to fight on, and to fight through. Dr. J. H. Abram, in a context of other significance,[1] describes two cases of gall-stone which he saw in consultation; the first patient had a sound heart and recovered; the other had a diseased heart, and died suddenly in the attack. And this, I think, is our experience in angina. Notwithstanding, in such an hour of trial, even in a young person with a heart presumably sound, we shall not be too confident; too often in such persons, and under such conditions, the results have been mortal. On consulting the authorities—such as Frerichs, and Rolleston—I find a considerable retardation of the pulse (50 or 40) in a gall-stone attack is not uncommon, especially at the outset. Frerichs has noted falls of rate to 28 and to 21. Weitzand[2] in such circumstances injected atropin, on which the pulse ran up to 120. Professor Saundby and I, after injecting atropin in a case of agonizing enterospasm, observed the same acceleration. On the other hand I have published a case of death in the agony of passing a gall-stone in a healthy young woman; at the necropsy the stone was found in the common duct, but the rest of the body, heart included, was perfectly healthy. Mr. Pinching of Gravesend, on seeing my published note, kindly informed me of a similar event in a woman æt. 57: the gall-stone had advanced one-third of the way towards the duodenum. No other cause of death was discovered. Conti[3] likewise has noted the reflex effect upon the heart of passing gall-stones. He dwells also on other subdiaphragmatic causes of cardiac syncope, and, accepting my hypothesis of inhibition as the cause of sudden death in angina pectoris, discusses a consequent "reflex palsy of the vagus," "with weakening heart,

[1] Abram, J. H., *Liverpool Med. Chir. Journ.*, March 1902.
[2] Weitzand, *Arch. f. exp. Path. u. Pharm.* Bd. xxxiv.
[3] Conti, *Riv. crit. di clinica medica*, 1904.

tachycardia, and even angina." On the other hand, he speaks also of vasoconstrictor conditions with dangerous rises of pressure. But he uses the name angina pectoris very loosely, and the whole article is rather vague and speculative. I am far from saying that death does not occur in the way Conti describes, but it is in the extremer stages of protracted angina; the usual course seems rather that the vagi fail by fatigue, then the accelerans gets the advantage; the beat rate quickens but the pressure and output fall, the heart dilates, and pulmonary œdema follows, often with such intensity as to become widely audible before dissolution.

I have alluded to Dr. Morison's interesting observation of the same inhibition in a case of angina pectoris with aortic regurgitation. While listening to his patient's heart during the attack, as the vagus inhibition subdued and expanded the ventricle, he heard the regurgitant murmur falling in intensity almost to disappearance. The pulmonary second sound, on the other hand, was enhanced, as in ordinary mitral disease. This observation disposed him to regard a diminution in calibre of the radial artery in angina as a syncopic rather than a constrictive change. This may be so occasionally: as we have seen vaso-constriction is not always present. Some sphygmographic tracings have shown the vagus effects on systolic contractions: these at first are slower, but unimpaired; then comes a fall of aortic pressure with more residual blood; then the help of a quicker rate until, as the heart recovers its balance, the output enlarges again, the rate falls, and after the relief the heart's vigour is restored.

I have alluded to the case of an insane woman in the West Riding Asylum at Wakefield who in a sudden paroxysm of fury staggered and fell dead. The heart was histologically sound, and death was put down to "dilatation" under the suffocation of fury; but subsequently I have suspected it was by inhibition, *i.e.* descent in diastole, larger contraction volume, and longer pause. Relaxation or contraction of the dead organ at the necropsy does not tell us much.

As regards the relation between the vagus and vasoconstriction, I will repeat the words I have already quoted from Professor Schäfer, who has told us that "vasoconstriction in the normal

state (*e.g.* under adrenalin) is stronger than vagus reduction; but that *the vagus influence may be deferred, and then suddenly show itself* (italics mine); when the auricles may stop, and the ventricular rhythm persist slowly and irregularly." [1] Thus in practice we find the vagus influences on the heart and the vasomotor function masking each other, while centripetal excitement of cerebral centres, indirect stimulus of the heart by rising blood pressure, and depressor function are in perplexed variation.

Now I have said more than once that in angina death is apt to occur on the first falter. It is not during the long battles that death is most imminent; on the contrary, patients come alive out of the most terrible attacks, or terrible successions of attacks. As Sir Lauder Brunton says,[2] "he is often arrested by no very severe pain." And Dr. Parkes Weber [3] testifies, "in angina pectoris it is often not the patients with the best marked attacks who die first." And we may add that if the patient dies during protracted seizures, he is liable to fall not on a climax, but at the outset of the particular sally which proves to be his last. If the heart be advanced in decay, some travelling bubble of wind, some sudden turn or emotion disturbs aortic pressure, the reflex trigger is touched and the heart drops to its rest. When such an event happens at the outset, it may be difficult, or impossible, to distinguish the death, as one of angina pectoris, from the lapse of ordinary cardiac syncope. In the fiercer assaults, death is often by way of ordinary heart failure.

There are on record, it is true, many cases of superficial or torpid suprasigmoid aortitis in which, as in many severe cases of syphilitic panaortitis, no anginal symptoms manifested themselves at any phase of the process. As we see in many an analogous impairment elsewhere, a very slow change may compass its own toleration; the nervous strands or endings may be not goaded but benumbed. Thus a strong man who, if suddenly throttled, would fight for life with demoniac energy, by very slow increments of cyanosis may sleep into dissolution. As in middle-aged, but still healthy and vigorous, cricketers

[1] Schäfer, E., *Brit. Med. Journ.*, June 6, 1908.
[2] Brunton, Lauder, *Encycl. Medica.*
[3] Weber, Parkes, *Lancet*, Jan. 18, 1908.

the consentaneity of eye and muscle becomes impaired, response by little degrees delayed, and little by little the component elements of function dissociated, rhythm more labile, and balance less automatic, until the sum of small increments becomes critical, so with years even in health disturbances are less and less readily neutralised or compensated. In a healthy state of the vessel the aortic reflex works well within the margin of safety, but we have seen how in a subinflammatory or irritable state this reflex becomes so exalted that each anginal seizure paves the way for an increasing frequency of reiteration; yet we still have to ask how far the integrity of the balance of the medullary centres is subject to, or susceptible of, higher control from above; and if in some cases we may have to suspect a morbid coefficient in centres superior to the bulb? The aortic tambour, a regulative apparatus working through its nervous endowments, not only governs systemic pressures, but also serves to control the pressure-constant for the bulb itself; so mutually dependent are these beautiful adjustments. For instance, in arteriosclerosis we may suppose a discord of function to be brought about in the cardiac centre by a deterioration of its own blood vessels; or by a fretfulness generated and perpetuated by summation of vexings of some tributary afferent surface, such as to drive it beyond control; or again by some loss of control from above. In conditions of this kind patients may drift into manifold states of peril; but these are obvious speculations which need no emphasis.

It was by considerations such as these that I was led to propose (at Yarmouth in 1894) that in the usual sudden death of angina pectoris the heart does not fail of its own intrinsic decay, but is stopped by reflex inhibition. Although a few physicians, Sir William Osler and Dr. Morison for instance, have mentioned this solution of the problem with approval, it is far as yet from general acceptation. Von Romberg, in the last edition of his book on the heart, makes no allusion to my view of the part of the vagus in death in angina; though he does suggest that it may be by shock—that is by paretic vasodilatation in the splanchnic area, an alternative which, as I have said above, may have to be borne in mind, but as an exceptional mode. Without by any means denying that in some instances death in angina may come

about by splanchnic shock, I remain of opinion that in the large majority the mode of vagus inhibition prevails. However the following case, taken from my private files, presumably illustrates the alternative of "shock," and the widely various susceptibility of individuals. "Mr. C. S. consulted me," so his family physician wrote, "for neurotic symptoms, especially pointing to vasomotor instability, with unequal vascular tone." When I came to examine his abdomen he proved to be very sensitive to taps just below the epigastrium. Of this he had been conscious for some years, so that he carefully avoided such taps or pressures, as they "made him feel very queer." His wife told me that one day thoughtlessly she gave him "a playful little backhander there" when he promptly fell down in a faint. This was a remarkable exaggeration of our common sensitiveness in this region; and in "abdominal angina" at any rate this may be one mode of death; but these cases are rare, and the opportunities of observing and verifying the precise way of death still fewer. The notion is current, and I think with some truth, that "sudden death runs in families"; if so, the tendency may consist in part in such hypersensitive reflexes. In his work on *La Mort subite* (1895) Professor Brouardel, among many cases of sudden death due to fright or other sudden emotion, narrates the following:—A labourer in full health was trying to lift a load, when another coming up to him said, "Get out, let a better man try," at the same time giving him a light blow on the stomach; the poor fellow dropped down dead immediately. No mark of violence was found at the necropsy. Of another man, he reports that in a fracas he was struck upon the testicles; he cried out, and fell dead. No lesion was found. Several other such verified cases he mentions also, in which sudden death followed blows on the epigastrium, larynx, and so forth; or sudden emotions, or injuries, too slight to be directly dangerous to life. A severe blow upon the chest may cause death by arrest of the heart; yet at the necropsy no disease be discoverable. Boinet says: "Chronic aortitis moreover may end in sudden death. Often the patient succumbs in a full attack of angina pectoris; more rarely he dies of a mere syncope, without appreciable cause, after some days of discomfort and precordial

pain. Potain and Rendu attribute it to an ischæmia of the coronaries, other authors suppose an anæmia of the bulb. Degeneration of the heart, alleged by Mauriac, seems improbable." Dr. Bence Jones' patient, whose case is narrated in my Cavendish Lecture (p. 147), died thus suddenly without appreciable cause in the heart. When the heart and its own arteries are diseased, the organ becomes unable to withstand a perturbation which a healthy heart would overcome; and when angina pectoris, even of some severity, has proved to be consistent with the prolongation of life for some years, or again when it has ended in recovery, we may guess that the factor of survival was a healthy, or comparatively healthy, cardiac mechanism able to resist extremes of reflex inhibition.

CHAPTER VII

DIAGNOSIS

In a full discussion of Symptoms and Causes Diagnosis has been so far anticipated that little may remain for a special section. For instance, in spite of what we are told about the difficulties of discrimination between angina pectoris and the host of spurious "vasovagal," hysterical, and other "functional" disorders, I venture to repeat that if the physician will concentrate his attention upon the definite features and processes of angina pectoris, not allowing his fancy to play fast and loose with spurious imitations of a malady so grave and so sinister, his difficulties will vanish. I cannot reiterate too tediously that time after time grave errors of diagnosis arise from temporising with angina, from watering it down on the supposition that there are gradations between the false and the real. What we have to decide is whether the patient be from first to last the subject of a turbulent disorder or of a mortal disease.[1] We are concerned with "trade and traffic in riddles and affairs of death." As Lancisi says of genuine and spurious aneurysms, no dissimilar contrast, it is of vital importance that, as with the Lydian stone, we shall be content with nothing less than the closest analysis. Of all maladies there are occasional cases which, at one interview at any rate, are hard to read; of angina pectoris such cases may now and then present themselves: for instance, as Nothnagel's vaso-motor series occasionally appears in true angina, there is a peril in too ready an assumption of spuriousness. Nevertheless I do

[1] Since these words were in type Lyman Greene, *Colorado Med.*, October 1912, writes: "I would especially emphasise the importance of minor anginas, and protest against the confused use of the term 'pseudo-angina.'"

not hesitate to say that in angina pectoris such moments of doubt are not more but less frequent than in other diseases of equal gravity. How much greater, perchance, may be the difficulty of arriving at a positive opinion—let us say in early cancer of the stomach, Addison's disease, tuberculosis, or typhoid fever. In initial stages, or even in later stages, if a disease manifests itself in some disguise, when for instance Graves' disease goes far without giving rise to either goitre or exophthalmos, or when in an old man a pneumonia or an effusion creeps on silently without giving rise to pain, cough, or obvious dyspnea —I need not multiply such examples,—under conditions so equivocal no doubt the physician, puzzled, or careless, may fall into error. In other cases, as Dr. Mitchell Bruce has pointed out, two or more possible sources of a case of angina may have to be unravelled. And when, under the influence of modern teaching, we confuse with unquestionable angina cases of spasmodic dyspnea, and cases of mere counterfeit, we may well lament that the diagnosis becomes difficult. Thus it is that some authors find themselves compelled to stipulate that there is no " true angina " without the radiating pains and the fear of death; but I repeat one might as well argue that epilepsy is not epilepsy unless aura, loss of consciousness, and convulsions be all present; or that Graves' disease cannot be said to exist until goitre, exophthalmos, rapid pulse and fine tremor have all put in their appearances together. Yet in such dilemmas, if we examine ourselves, have we not too often to confess that when we erred, or vacillated, it was for lack of thoroughness, of accuracy of observation, or of perception of the relative values of the signs and symptoms *inter se*? But in the inquest of angina pectoris no soft options are tolerable. Curschmann, who took a naïve pride in his unquestionable powers of diagnosis, described a certain case of spurious angina [1] as one which agreed in all respects with the genuine disease (*alle Symptome wie bei echter Angina*). These symptoms were pain and tightness *at the heart, severe* (heftiges) *palpitation, oppression to the degree of syncope*, radiating pain in the left arm (italics mine). Now I may ask any experienced physician if this group of symptoms is characteristically that

[1] Curschmann, *Lehrbuch d. Nervenkrankheiten*, 1909, p. 832.

of genuine angina? Indeed the author himself proceeds to say that, as they occurred in a hysterical young woman without syphilis, he was sure of the true diagnosis. Of course he was; then why worry about it? The chief difficulty is, as Rauzier says,[1] to recognise at times the partial dependence of such symptoms on organic disease; as in my case of the housemaid with aortic stenosis (p. 189). Rauzier publishes two such cases, and wonders if the pain and distress had in them a core of true angina or not. Now both presented a double aortic murmur.

Too well aware of the black list of my own errors, or shortcomings, in clinical diagnosis, I venture notwithstanding, "ut homunculus unus e multis," to feel some assurance that under the head of angina pectoris we ought very rarely to go far astray. Cases of difficulty, of suspense, there are, as we shall see; but these equivocal instances are not of the counterfeit class, and cautious effort tests, during tranquil intervals, are generally crucial. However, *among the spurious cases* I may refer to a kind mentioned by Sir William Osler[2] in his Lumleian Lectures which may be very equivocal. Certain highly susceptible persons, if brought face to face with a case of angina, and constrained by anxiety and sympathy, may, under some potency of suggestion, reproduce in themselves the features of the disease, as it were in a glass. That the counterfeit may be very close the author's cases prove to us. I have not had this experience; but I remember such a "suggestible" person, highly strung under the trial of our final M.B. examinations, who, when questioned by a physician concerning asthma, extemporised an attack of this disease; and on another day when questioned by a surgeon on renal colic threw himself into this attitude of suffering. The examiners, not without excuse, resented what they supposed to be mockery; and I, who knew my man, had some difficulty in appeasing their wrath. Thus in a neurotic patient of either sex, especially if near or under middle age, it may be difficult, at one interview, to discount some extravagances of blood pressure and a thoracic oppression due only to "nervousness or vasomotor storms."

[1] Rauzier, *Province méd.*, Feb. 3, 1912; quoted *Arch. des mal. du cœur*, Sept. 1912.

[2] Osler, W., Lect. II., *Lancet*, March 26, 1910, pp. 839-840.

I must content myself here with repeating axiomatically that angina pectoris after middle life is usually atheromatous; under the age of 45, if not the mock disease, it is usually either syphilis, rheumatic fever, or influenza.

Concerning the touch of *the minor form* of the malady, as in latent epilepsy so in angina, the story of the attacks may be so unskilfully narrated, so defective in data, so casually alluded to, or so overlaid by other and more imposing disorders, as to escape even the attentive medical critic; yet such cases should cause no more than transient hesitation. For instance, in the case of a certain middle-aged lady I felt some hesitation, until it came out that the patient had twice been awakened in the night (4 or 5 A.M.) by substernal pain and oppression, without dyspnea, compelling her to sit up in bed; this was almost though not quite decisive. Rarely can it happen that a broad and discriminating review of all the facts would fail to build up a decisive opinion: a review of the patient's age; of his temperament; of the state of his tissues and especially of his vessels and aortic mechanism—as tested for instance by the dimensions of the aorta, the position of the subclavian and innominate, the quality of the second sound, and the effect of exercises in calling out, or failing to call out, a systolic murmur at the base; the persistent range of his blood pressure; in a patient of or under middle age the possibility, or even the stigmata, of syphilis—such as scars, old iritis or choroiditis, Robertson's pupil, loss of knee-jerks, Wassermann's reaction, or the history of some other toxic cause of aortitis, such as influenza; a startled check or warning grip as contrasted with a gasp and sob as of a cold douche; or again a coherent story with constancy of symptoms as contrasted with a flustering story of incoherent complaints.

Perhaps of my own list one of the more difficult cases to analyse was this:

Miss E. S., æt. 35. Has had much worry; two and a half years ago she fainted away. Then, and for some time after, had attacks of severe pain in the side, a hand's-breadth below the breast (7th-8th rib). Then the pain moved to the left arm, with a suffocative feeling and panting. "Always having it." (A "suffocative feeling" and panting are more suggestive of the so-called false

angina.) The pain starts at the inner left wrist, running up the inner forearm; sometimes it is at the anterior axillary bend of the fifth rib, sometimes in the lower intercostal region. During and after the attacks there is severe hyperæsthesia of the whole left arm and thorax. The attacks are quickly relieved by amyl nitrite. They are never brought on by exertion, but follow annoyance or fatigue. When free from attacks, can walk up stairs or hills. No anæmia. The systolic pressure rose to 140, but probably 10-15 degrees due to nervousness (we had not time to await the calmest moment). Heart and vessels all seemed normal. No neuritic or neuralgic "points." The patient was quickly cured by change of scene, judicious hydropathic methods, and strong suggestion. There was no history of influenza or other infection; syphilis was inconceivable, and no signs of it were to be detected.

Pains, heaviness or numbness, or combinations of these in the left arm are quite commonly associated in neurotic women with the still better known submammary pain. Vasomotor false angina is often relieved by nitrites, especially by amyl nitrite, though not so regularly as we might have expected.

Dr. Byrom Bramwell[1] tells us he has seen cases in young persons in which, had they been older, the diagnosis of angina pectoris would assuredly have been made; but in which complete and lasting recovery (and the age) "showed that the angina was functional." For, he adds, "it was impossible to differentiate functional and organic forms." My distinguished friend does himself an injustice; no one is better able to discern these vital distinctions; but his judgment, if I dare say so, may have been warped by some systematic prepossession? In the study of angina I am disposed to say of not a few of my distinguished colleagues, as was said of Patmore, "that he did not so much follow the light, as plant the light where he chose." I have shown, I hope, that in young cases, syphilitic or otherwise infective, angina pectoris often does pass away.

As an instance of differential interpretation of pains, I will call attention again to Dr. James's case (p. 442).

It is often hard in aortic disease of the heart to say whether some pains are of angina or not. If not, if they be due to fagged heart, the pain is more about the lower area of the side, but is apt to rise into the left upper breast; a pain of

[1] Bramwell, Byrom, *Brit. Med. Journ.*, Jan. 15, 1910.

cardiac distress does not seat itself under the sternum. Extrasystolic leaps and bounds of the more violent degrees sometimes cause pain about the heart, but, so far, the sign is rather good than bad.

Then there may be, and, if so, more significantly in the stealthier cases, the peculiar sense of arrest without dyspnea (we have seen that if there be dyspnea the heart or lungs will prove to be diseased also); *the unaccountable touch of warning*, something less perhaps than a *meditatio mortis*, but yet significant or sinister out of proportion to the degree or duration of illness, a feeling in my observation quite alien from the conflicts, desperate as they may be, with paroxysms of asthma, of cardiac or renal dyspnea, of laryngeal spasm, of pulmonary infarction, of tabetic crisis, or "nervous crisis," and so forth—encounters in which the patient may win or lose, but which at worst are not an instinctive horror but a rational alarm, a battle with an open enemy. That ominous touch it is which often speaks to the wise just when a hint is wanted; namely in those light, swiftly transient, early apparitions of angina whose interpretation may not be clear; the touch of the tipstaff, the sense of an uncanny call, is frequently there. I have cited the case of a patient who suffered from anginal pain in the centre of the hand (see p. 293); in that case sure diagnosis was perhaps at first impossible, but the patient's own foreboding was true enough.

Pain then seated in the area commonly called "cardiac" or "precordial" is, as we have agreed, a common sign of cardiac disorder—valvular or functional; but neither in seat, nor character, nor aggravations does it resemble angina pectoris. I have quoted Dr. Mackenzie's contrast of this "precordial pain," dull and persistent if at times rather paroxysmal, with that of angina. This lower intercostal pain may however strike up the left side of the chest into the neck. Intolerance of pressure or percussion in this affection is more conspicuous than in angina; it is most conspicuous perhaps in functional cases.

In another case, recently under my notice, the pain was again the principal feature, not only in the diagnosis, but in the patient's description; in the earlier attacks the sensation in the arms

was rather one of discomfort and weakness than of pain. This sensation first came on after long and enthusiastic dancing, when he felt much exhausted. The discomfort of the arms extended "down the flesh of both arms down to the wrists." In later attacks the arms became exceedingly painful, the pain being now brought on by ascents, though occasionally it would come on during rest. One or two of his worst seizures awoke him soon after midnight, the pain in the arms, and latterly down to the fingers, being "terrible." On ascents or other exertion the pain or distress was entirely in the arms, and his description was of something more distressing than ordinary pain. On examination, which as the chest was fat was not very easy, the cardiac impulse in its normal site, even in the prone position of the patient, was perceived with difficulty, and the first sound wanted quality. Percussion was negative. Nothing seemed definitely wrong about the base of the heart or the aorta. He had lived rather freely and had a very anxious business; he smoked, but not to excess; tobacco did not seem to be the cause. He denied syphilis, but I have blamed myself for not insisting on a specific course of treatment. I have said that when he saw me the pain in the arms was the predominant, if not the only, symptom, yet some attacks—and, oddly enough, the earlier ones—had been attended by an "exhaustion" in the chest; in the later, either the "chest symptoms" had passed off or were lost in the agony of the arms; although again it is remarkable that "a sense of something more than mere neuralgia or rheumatism" was borne in upon him. Not long afterwards the malady took on the usual form, and a year or two later he died in a seizure. Another patient tells me, in spite of some dubious reassurances of mine, that the pain from which he suffers is no ordinary pain; and, as time goes on, I am more and more convinced that he also is right. Yet his heart to all direct appreciation is healthy, and once during an attack of the pain presented, to my perception at any rate, no aberrancy. Now however the patient is checked while walking, and often has to sit down, or lean against some support. And I note that he walks by preference along a road where there are seats.

Even concerning the common radiating pains, error is not out of the question; generally by imputation of angina to

intercostal neuralgia which, as we have seen, may be paroxysmal, and attended with much nervous perturbation and with cardiac disorder; or to the more tenacious pains originating in certain intrathoracic (especially aneurysm) or spinal diseases, for which however we have the tests of painful spots, the more continuous duration, the comparative independence of bodily and emotional movement, or signs of other meaning, such as those of mediastinal growth, and so forth. In such cases the X-ray picture would come to our aid. In an aberrant case, or in one of angina minor, the response of the pain or oppression to ascent, even a gentle acclivity, often affords a crucial point of diagnosis. Dr. Dockray's patient (p. 294) could move about the house without pain; he could even turn the winch of his printing machine with impunity, which was curious; but to walk up even a moderate ascent, especially if soon after a meal, pulled him up at once and inexorably.

On the other hand, it is unquestionably true that in many cases, and in many attacks, of angina, only too genuine and severe in kind, even in a status anginosus, there may be no death omen. However this symptom is one for which, if not spontaneously mentioned to me, I am rather reluctant to enquire.

Parry, in his descriptions of angina pectoris, dwelt rather too much upon syncopic symptoms, and so was led to propose the name of Syncope anginosa. Still there are cases no doubt in which, especially if the attack be known only by description, there might be some hesitation between angina, petit mal, a Stokes-Adams syncope, or an ordinary fainting fit.

The more eccentric seats or courses of anginal pains I have indicated already; the practised observer is generally prepared for them, partly by their behaviour, partly by their association with some warning; and I must not reiterate more than is convenient (*vide* p. 296). Rendu[1] has published a good example:

A man, æt. 40, of robust appearance, was attacked by influenza. After this he began to suffer from peculiar attacks, consisting in a kind of crisis arising in the little finger, extending up the arm, and finally becoming more marked, accompanied by feeling of oppression

[1] Rendu, *Journ. de méd.*, June 10, 1896.

in the chest and agony, with, on some occasions, syncope. After a short period in hospital these attacks became somewhat better, but then for a while recurred daily. There were no physical signs of disease, either of heart or lung.

In discussing the diagnosis in this case, whether it were a case of angina pectoris or not, Rendu was rather inclined to withhold the diagnosis of true angina pectoris, because there were no signs of cardiac affection; because he supposed angina to be very rare before the age of 40, and because the pain began peripherally. The previous history of influenza was also, in his opinion, in favour of a "neurosis." The case ended in recovery. I think Rendu was clearly wrong, in any case his three reasons were insufficient. The case was published not long after the commencement of the virulent outbreak of influenza at the end of the last century, before its occasional attack upon the aorta was recognised (p. 268). A few years later Rendu would probably have come to a different diagnosis; for he has done good service since in describing the aortitis and its symptoms which may follow rheumatic fever (*vide* Aortitis, p. 155).

Neuralgia of the phrenic nerve might cause some suspicion of angina; in one case of diaphragmatic pleurisy in an elderly man I was for a while in doubt. Phrenic nerve pain is referred to the insertion of the diaphragm into the costal arch, passes upwards on the border of the sternum, and between the scaleni in the neck; the respiration is cramped and shallow, and racking cough, hiccup, and even vomiting may appear.

Of *physical signs* I say nothing; often there is none (p. 345). Charles Sumner was examined by many leading physicians in the States and in Europe, and in every case with negative results. Perhaps the area of the aorta was not carefully mapped out. But the not infrequent association of angina pectoris with *aneurysm* must not be forgotten; in the presence of both to rest satisfied with a diagnosis of either would be unworkmanlike. In such cases pressure signs, radiography, and other means may assist us to a complete diagnosis. The spinal pains of a posterior aortic aneurysm are more continuous than those of angina; at first they are little more than a weary ache, and are usually alleviated by changes of posture. But aortic aneurysms in the supravalvular area,

associated with angina or not, may escape the most rigid scrutiny, even by the X-rays. We must never forget that diseases may run in company. I may make a reticent allusion to the report of a certain well-known case of "gastric ulcer simulating angina pectoris." The explanations of the pain by a perigastritis affecting the solar plexus, splanchnics, and dorsal nerves were quite ingenious; but then it came out that a short, soft murmur of aortic regurgitation had been heard! We have seen that many of the older writers swept under the title of angina a motley collection of cases of "gout of the heart," "gout of the stomach," mesenteric arterial thrombosis, cardiac and pulmonary embolisms, acute pancreatitis, and so forth; as pathology then was they could hardly avoid the confusion: we must try to do better.

A right interpretation of the uneasiness of *abdominal angina*, even if the physician be on the alert to recognise the characters of this form of the malady, may for a time be difficult. I think in the following case the diagnosis of "typical abdominal angina" was questionable, that its symptoms belong rather to the well-known group of mesenteric atherosclerosis with consequent atrophic and other disorder of the bowels (p. 310).

Female, æt. 42, suffered for years from crampy recurrent pains of colicky nature. Atherosclerosis present in high degree. Blood pressure enormously high, heart enlarged. Died of an apoplectic stroke. P.M.: Extreme sclerosis of abdominal aorta and its chief branches. Kidneys "intact." [1]

Tobacco-smokers, gouty persons, coffee-drinkers, and some nervous persons are liable, especially in later life, to attacks of cardiac irregularity, especially during and after meals, with epigastric distension, oppression, or even pain, a series which, with some degree of arteriosclerosis in radial artery and aorta, may simulate *epigastric angina* very closely. Ortner says (*loc. cit.*), and I agree with him, that it is not sufficiently realised that a race of extra systoles may cause oppressive pains, often substernal or epigastric, and sense of tightness. These are the riddles which call for all our discernment, and a diagnosis on one visit may be scarcely possible; but in a few days the

[1] Rossbach, *Münch. med. Wochenschr.*, 1910, No. 19.

recurrences of arrhythmia during rest, and without the epigastric pain, the comparative ease, when the stomach is empty, of walking slowly even uphill, the ability, even at worst, still to go forward, if with some discomfort, the effects of diet and remedies and so forth, will enable us, after some care and watching, to come to a decision. Eructations of wind mean little in this diagnosis; they accompany all such conditions. In one ambiguous case of this kind I was assisted to the correct diagnosis by a note of Broadbent's, which I have found useful in not a few cases — that cardiac arrhythmias, of the extrasystolic kind, coming on at rest after getting into bed, are of functional origin. It has been pointed out that a globus of wind, or spasm in the œsophagus or at the cardiac orifice of the stomach, may simulate slight anginal attacks. Mr. Verdon says it is more than a simulation (p. 344). Dr. Hertz, in his interesting "Goulstonian Lectures," [1] described this kind of discomfort and fulness under the sternum in the middle line as caused by rapid inflation of the small balloon introduced for experimental purposes into the gullet. However, speaking very generally, obvious perturbation of the heart, palpitation, arrhythmia, or even acceleration is against a diagnosis of angina. On the other hand, a "gastric or abdominal case," whether a true angina or not, is by no means safe; and such patients, usually elderly men with abdominal and other arteriosclerosis, should be warned against exertion after meals, or while dyspeptic or flatulent; and we shall not forget that gouty plethoric persons are apt subjects for angina.

Akin [2] cites this case, of a man, æt. 56; gouty, and a large feeder. On walking after full meals would feel an oppression, and a little pain under the ensiform cartilage. *Also was short of breath.* He would wait, belch, and then go forward. Pulse 90, tense. Aortic second sound accented. Urine contained a little albumin, but no casts. So far the case may not have been anginous, such symptoms are common in disordered hearts; yet at a later date angina supervened. Here the dyspnea might have thrown the physician off the diagnosis.

At the Belfast Meeting of the British Medical Association a case of sudden (cardiac) death was reported in a young man per-

[1] Hertz, *Lancet*, May 6, 1911.
[2] Akin, *Journ. Amer. Med. Assoc.*; Rpt. *Lancet*, June 20, 1909.

forming some feat of athletic emulation. At the post-mortem no disease was discovered, but his stomach was found crammed with cherries. I believe the line of interpretation of such cases, as of the so-called angiospastic or angiosclerotic abdominal pains, will lie towards tension upon the mesenteric and other visceral investments and connections. These tracts are very rich in Pacinian bodies. In one such case these bodies were found diseased.[1] And in old persons, as Dr. Hugh Anderson's experiments (p. 475) show, even in cases in which no definite degenerative changes may be demonstrable, the heart is far more touchy; so that death on mesenteric tension is not surprising. Lastly, thrombosis of a coronary branch might occasionally, as Pal says, simulate the abdominal form of angina rather closely (*vide* p. 311); the pains, if any, may strike into the left arm. Or the collapse, *continuous* epigastric or lateral pain, oppression, and rapid irregular pulse may simulate a visceral perforation (see case, p. 451). I will conclude this paragraph with short notes of the following case, in which my own diagnosis was at fault.

An active and healthy elderly man was walking quickly to keep an appointment, when he was startled by a distressing sensation in the epigastrium, " as if a half-brick had rolled over in his stomach " ; he pulled up, and then walked slowly to his meeting, and thought no more of the matter. For some months before, however, he had noticed a disposition to intermittences of the heart; doublets, triplets, quadruplets, and so on; or with more arrhythmia : and for the first time he had had a slight attack of gout. Eight or ten weeks later he took a long cycle ride in a hilly country, and much enjoyed it; the next day he walked some hours on the moors, and returned fatigued; but after a rather full tea, started for the railway station, a mile distant and uphill. Almost at once he felt again the same oppression in the epigastrium; this returned incessantly as he walked, so that he reached the station with difficulty. He was directed to live carefully, and to avoid exertion; still the oppression would return on slight efforts. In London he could not manage to walk from the steps in Waterloo Place to Piccadilly Circus without bringing on at least a touch of it. One day he was unexpectedly called upon to walk up a short, steep rise to a house where he was to call; as he cautiously began to ascend a pain seemed to rise under the sternum, and to spread over the front of the chest; it was not severe, and was unattended

[1] Case quoted, Hüber, C., " Sensory Nerve Fibres in Viscera," *Journ. Comp. Neurol.*, 1900.

with inward dread, only with rational apprehension. It returned a little as he essayed to walk on, still he managed to reach the house, and felt no more of it. The diagnosis of angina seemed then inevitable; he was put under strict rest and diet, and some anti-gouty remedies were administered. In a few weeks the whole trouble vanished; and gradually the patient returned with impunity to the active life which some years later he still continues. The malady proved to be no more than may be indicated by such a title as "gouty dyspepsia"; and the pain in the expanding thorax seems to have been but a part of other muscular pains about the chest, back, and loins, which likewise were interpreted as "gouty." (Compare case, p. 259.)

There is not much fear of error between "abdominal angina" and abdominal aneurysm. Dr. Nixon [1] read for me notes of 179 cases of abdominal aneurysm; in none was the pain anginoid; even when paroxysmal it was more after the kind of renal or other colic, or radiating down some nerve, such as the sciatic. Generally it was a boring pain, often severe; the seat usually lumbar. Death was practically always by rupture, when the pain was indeed more after the manner of angina pectoris.

To discriminate *tabes*, whether in abdominal or thoracic angina pectoris, as an alternative, or associated disease, will rarely be difficult; but we must remember that in uncomplicated syphilitic angina the pupils may have ceased to react to light, or the patellar and Achilles' tendons to the tap. In one of my cases of syphilitic angina without tabes, the light-stop sign was present. There are many differential signs by which the diagnosis may be made, and the "crisis" of tabes distinguished; but we shall never forget that aortic disease, due to the same virus, often brings about a combination of angina and tabes, with a possible aneurysm in addition. It is conceivable of course that upon some hapless sufferer both an anginous and a tabetic crisis may fall together. So far as my small experience of such mixed cases goes, the paroxysmal "gastric" pain of tabes itself is intercostal, not substernal in seat; and, if the heart be disturbed, it is rather by palpitations, which are not characteristic of angina pectoris. The pain of "gastric crisis" usually ranges about the area of the 7th, 8th, and 9th dorsal nerves, but we know of no reason why tabes should not attack the same spinal

[1] Nixon, *St. Barthol. Reports*, vol. xlvii.

segments as those concerned in angina, and might thus set up the same referred symptoms. Some such interpretation might hold for a case of tabes published by Vulpian,[1] in which, although angina pectoris was diagnosed, at the necropsy, the heart and aorta proved to be normal. A peripheral neuritis of cardiac nerves is alleged by many writers on nervous diseases (Oppenheim, Pitres, Vaillard), but with little or no definite evidence.

The experience of a particular case reminds me that the possibility of confusion between an attack of angina minor, or sine dolore, and petit mal should be mentioned, as contingent vasovagal or other circumstances might make a single attack a little difficult to unravel. Still, with both alternatives present to the mind, the diagnosis could not remain long in doubt.

The factor of tobacco cannot be tested except under a rigid abstinence of at least six weeks. I have suggested that in tobacco cases some cardiac arrhythmia is always (?) present (p. 247). Some cases attributed to tobacco were probably infectious angina.

Appendicitis seems to be capable of anything; but embolism of the mesentery, pancreatitis, gall-stones, plumbic, neurotic, and other belly-aches, are very unlikely to simulate even abdominal angina. Still there are cases, such as one reported by Ortner,[2] and another by Stengel,[3] out of 25 cases quoted from Max Bruch of Vienna, in which atheroma (probably with thrombosis ?) of the mesenteric vessels, with cramp-like pains, colic, tympany, and constipation, simulated the abdominal form of angina pectoris.

Here, in its formal place, the radical distinction between angina pectoris and *paroxysmal* cardiac or uræmic *dyspnea* must again be emphasised. Although to confuse these states seems scarcely venial, yet among records of angina pectoris we may continually detect admixtures of cardiac cases, of pulmonary œdema or infarction, and of high-pressure dyspnea. Not long ago I read these published words of a justly esteemed teacher, " A man walking uphill one day was seized with sudden breathlessness, so that he had to sit down, in fact he had a very bad attack of angina pectoris " ; well, so it may have been, but

[1] Vulpian, *Rev. de méd.*, 1885.

[2] Ortner, Volkmann's *Sammlung.*

[3] Stengel, Toronto Meeting Brit. Med. Assoc., 1906; *vide Lancet*, Sept. 15, 1906.

the "fact" of dyspnea was a contrary fact. Probably the case was just heart disease; at any rate it was complicated with heart disease. On the paroxysmal dyspneas of renal disease, and of Hyperpiesia, I have written at length (pp. 315-316. Compare also Elliott's Equivocal Case II., p. 447). I can only repeat that the assumption of the name of "angina pectoris," or of "angina sine dolore," for these seizures is without excuse.

With Mr. Harris of Ely I once saw a man, of 79 years of age, in whom attacks of anguish led to a suspicion of angina pectoris ("sine dolore"). The heart was arrhythmic, and the auricle probably fibrillating. The physical signs suggested myocardial degeneration. The "fear of death" was a part of a heavy depression of spirits, and we decided that the case was not one of angina, but of heart disease and melancholia. He improved for a time, but at a later date died in the ordinary course of heart failure.

The following case is a good instance, I can scarcely say of the difficulty of diagnosis, for in my opinion there was none, but of divergence of medical opinion; divergence engendered of the looseness of phrase in respect of angina pectoris. A man, about 60 years of age, of despondent but not unhealthy aspect, complained of pain in the region of the heart. He laid his hand upon the submammary region of the left side. The pain, though variable, was not paroxysmal; it came and went in a capricious way, observing no moments of exertion or emotion. At some hours of the day or night, however quiescent the patient, it would appear; as at other hours it would recede. Though he complained of it with morbid importunity, for he was convinced it was due to disease of the heart, and would kill him, yet there was no organic horror. The site, character, and behaviour of the pain, the temperament of the patient himself, and the other symptoms all pointed to hypochondriasis. However, as ill-luck would have it, there was a systolic murmur at the apex of his heart, a murmur unaccompanied by any signs of cardiac alteration, or of irregular distribution of the blood, and due no doubt to sclerosis. He could lie down, and continually would lie, curled up on bed or couch without the slightest embarrassment of the breathing; and, in his dejected way, could walk up and down stairs, or out of doors, without

renewal of his discomforts. Nevertheless he was pronounced by two physicians to be suffering from angina pectoris. He was sent to me by a third physician, a man of large and ripe experience, who demurred to the diagnosis. I also assured the patient that his case was neither one of angina nor of any active cardiac disease; there were no signs of circulatory disorder, and the pain had neither the seat nor the manner of anginal pain. The patient, disappointed by the mildness of our diagnosis, went off to yet another consultant, who promptly accepted the more dignified anginal interpretation. The patient threw up his business, the worst step he could have taken; and, as I heard more than once at later dates, succumbed, not to angina, nor any mortal affection, but to hypochondriac invalidism. Such are the consequences of the parrot-like repetition that anginal pain is "in the precordial region." We repeat it till it imposes itself upon us as truth.

Another interesting example of a like error was narrated a few years ago by Dr. Shadwell,[1] in a discussion at which I had been describing some cases of influenzal angina. A lady patient, three days ill of influenza, was sitting up in bed *gasping for breath*, she became "livid" (not pale), "her hands and feet were cold." At first Dr. Shadwell thought these attacks "remarkably like those of angina pectoris"; but, as a good diagnostician, he proceeded to detect and to verify certain tender points in the course of one of the intercostal nerves on the left side and on the spine, and finally discerned that the case was one of intercostal neuralgia. Dr. Shadwell assured us that his case, which was one of the spurious kind with intercostal neuralgia especially apt to arise in women after influenza and similar enfeebling maladies, might well have been taken for angina pectoris. Dr. Shadwell was probably right, yet he might have hesitated before the evidence, since firmly based upon clinical and pathological records, of genuine angina pectoris as no very rare sequel of influenza. If the pain is precordial, if the patient lays his hand under the breast and across the apex of the heart, the heart may perhaps be affected, even gravely affected; but the presumption so far is against angina pectoris. The pain may be "referred" from some other viscus, or be a "mere neuralgia."

[1] Shadwell, *Brit. Med. Journ.*, May 13, 1905.

Intercostal and other neuralgias are not definitely aroused by movement as are anginal pains. They are more "spontaneous," more capricious, more wearisome.

Dr. Head and Mr. Mummery have pointed out that pain radiating downward to the shoulder and arm, and even paralysing the arm and hand, may spring from so eccentric a source as a lower back tooth. Balfour recorded an alarming attack in an elderly gentleman of "cardiac pain" with failing and accelerating pulse, which happily resolved itself into an attack of shingles.

Coffee, tea, and tobacco poisoning will not be forgotten; doubts of this kind, as I have said, can be cleared up only by a six or eight weeks' abstinence from the suspected article. The tobacco oppression does not arrest the patient with the same imperiousness, he can walk on, indeed he is often restless or agitated; still it may be impossible in a middle-aged man with some arteriosclerosis to come to a decision until tobacco has been laid absolutely aside for six weeks. For occasionally, in its slighter degrees, when the cardiac arrhythmia rarely absent in tobacco cases may be scarcely notable, a substernal oppression hard to distinguish from angina minor may occur. Sir W. Osler[1] says that tobacco angina may prove mortal; my own impression is that not a few cases of so-called "tobacco angina" are angina proper; *e.g.* of syphilitic or otherwise infective origin, and that the mortal cases are of them.

On the subject of *spurious "anginas"* perhaps more than enough has been said (Chap. II.); I will not refrain from repeating Balfour's sarcasm that the name "pseudo-angina" is useful only "to hedge the diagnosis." Not that the false anginas are always "hysteria" or "temper." The criterions formulated by Huchard, though too formal, may be useful in practice; the diagnosis lies not in this item or in that, but in the sum of the series. We have seen that agitation is not unknown in cases of angina only too genuine, the patient may even pace the room, and show other movements of distress (p. 328). We have all seen such patients quickly sit up in bed, or even spring to the floor. One of my patients in his worst moments rolled in the bed to and fro, swaying his arms and moaning; but attacks so cruel as to break through the awe

[1] Osler, Sir W., *Lancet*, April 9, 1910. See however p. 246.

of the disease are, I think, always on other grounds indubitable. The pain or oppression of the "Vasomotor" cases is often severe, but the pain is commonly in the lower intercostal area, with precordial anxiety and hyperæsthesia, and attended with sighs and pantings. Or it may occupy the upper chest, usually the whole upper breast; it is more diffused, not definitely sternal, the whole thorax is bursting; and in the arm the pain, if any, may not observe the ulnar or any definite district. The hyperæsthesia of angina pectoris, according to Mackenzie, is deeper, and may be elicited in the absence of complaint by pinching up a muscle—pectoral, sterno-mastoid, or trapezius. I have seen superficial skin tenderness now and then soon after an attack of angina, in one case very distinctly. Quickened pulse, palpitation, or arterial throbbings suggest "neurosis." Sometimes the mock angina in its returns of distension, or fulness with panting, observes a periodicity which is not seen in the briefer apparitions of the true disease. In most cases the eye, temperament, and history of the patient, and the state of the arterial tree, are decisive. Still vasomotor oscillations often appear in genuine cases, perhaps in few are wholly absent; moreover such phenomena may precede and give warning of a genuine seizure, and such a double process in an elderly woman might for a while be difficult to disentangle. Polyuria is a feature of both maladies. It is not my experience that spurious angina brings about arteriosclerosis. I once saw a case—with Mr. Cory of Soham—of very slowly established pulmonary thrombosis after pneumonia in which for about a week the pallor, oppression, and fear might for a moment have suggested angina sine dolore; but the more or less continuous course of the incident, the hampered respiration, and other features were distinctive; although until a few seconds before the sudden death the pulse was regular at 84, and normal in quality. Of my own experience, I must repeat that, although I have been in the way of seeing a good deal both of cardio-arterial and of nervous disorders, I cannot recall any case of the so-called "pseudo-angina" which could have deceived a careful observer, in possession of all the data, unless it were perhaps one of the rarer tobacco cases, or possibly one of arteriosclerosis or aortic disease with a fringe of neurosis but no angina.

It would be unbecoming in me, however, to assert on my limited personal experience that there is never room for wavering between " organic " and " functional " cases. For instance, some years ago I heard to my regret that a gentleman well known to me, at any rate by reputation, had been obliged in the prime of life to retire from an important position on account of angina pectoris. He had access to the best medical advice, and no doubt availed himself of it, and acted upon it. A few years later I learned that after an interval of complete rest he had entered again upon an active life, and was in good health. I wrote therefore to a common friend of his and mine, an eminent physician, now taken from us, to enquire if the case had been one of angina pectoris, perhaps an infection, ending in recovery. Our friend, who had been consulted in the case, replied to the effect that he had found " no evidence of any infection, nor indeed of arterial disease, nor of high blood pressure; but rather of atony," and that he himself had opposed the verdict of angina. His opinion at the time was that " the case was one of overwork, neurasthenia, and perverted metabolism." We may not unreasonably suspect that even in this case also, difficult as it may have been, the diagnosis of angina pectoris was not only wrong but not justifiable on the facts. Still genuine angina may occur in neurotic subjects (*vide* Case 241), with epiphenomena which might divert the observer from the fundamental diagnosis. This lamentable mistake has occurred more than once within the circle of my own experience.

The stealthy growth of *aneurysm*, of *adenomatous*, *syphilitic*, or *other neoplasms* of the mediastinum, pleuro-pericardial fibrosis, and so forth, must not be overlooked; in syphilitic cases aneurysm and mediastinal gumma will not be forgotten: but this is not the place to enter into so large a digression. We must make sure that angina is not associated with a latent aneurysm; pressure pain may persist between anginal attacks. In all such cases the X-rays will be used. I have mentioned already one case of angina in which dilatation of the aorta was thus definitely verified; physical signs had indeed indicated it. The second case was a remarkable one in this respect, that the first examination " on a bad day " likewise clearly showed dilatation of the aorta; but on a second occasion, when the patient had felt

much better, the vessel had recovered its normal dimensions. I remember one case of mediastinal growth, and one of extensive fibrosis, in both of which angina was at first suspected, but set aside in part by the ray picture. I have remarked incidentally concerning perforations of heart or stomach, pancreatic hæmorrhage, etc., that if for a moment they might be taken for anginous attacks, the collapse, and in many of them vomiting, would soon remove them from this category.

Some authors approximate *Stokes-Adams disease* to angina pectoris, but by confusion rather than by comparison. I have no recollection of a case of my own partaking in any considerable degree of both diseases, though of course the double event is not unlikely, especially in syphilis, influenza, or rheumatic fever; and the a.-c. interval may for a while be prolonged. In the syncopic variety attacks of epigastric anxiety, pallor, and suspension of respiration might at first suggest epigastric angina, or angina sine dolore; but the effects of atropine, the venous pulse, and so forth, should soon clear up any doubt. I will conclude this somewhat critical but, I trust, not censorious section, by taking to myself a confession of Dr. Goodhart,[1] a touch which makes all physicians kin; he says, "I sadly and sorrowfully admit it. I have made mistakes so often that I no longer blush for them." Few of us perhaps can so well afford this frankness; none in a few words could have set us a better ethical example.

The effects of nitrite of amyl, positive or negative, have been put forward as a diagnostic test. It is not worth much; it relieves many spasmodic conditions not angina pectoris.

[1] Goodhart, Sir J., *Lancet*, July 1, 1905.

CHAPTER VIII

PROGNOSIS

Prognosis, even more than diagnosis, is almost wholly implied in the discussion of symptoms; yet here again I would formally declare that *the issue of angina pectoris is by no means always mortal.* If the interpretation of its symptoms be such as I have advocated, we have seen that death in angina pectoris is an accident, in old persons a frequent accident, yet, even were this issue invariable, it is a contingency nevertheless. The proportion of cured cases on my list is large, and includes a few elderly persons with the disease in its classical form. Dr. James Mackenzie reports that one of his patients, a man æt. 48, with all the classical symptoms of the disease, recovered. Broadbent [1] described a case in a man æt. 50 (apparently syphilitic ?), in which, but for an aortic murmur, the patient got well, and returned to business. "Fortunately," as he surmised, "the coronarics were not implicated." His diagnosis was of angina nevertheless. One of Fothergill's patients, a typical case, with the usual breast and arm pains, æt. 30 (syphilis ?), recovered completely, and was in good health twenty years later. Fothergill gave him cinnabar and Bath waters. Yet among medical men the conviction that angina must be mortal is so strong that if it fail to kill it is deprived of its name. I have quoted Broadbent's remark that the dead house is no place to look for cures. Desportes, surveying his collection of cases, concluded that a favourable issue might be hoped for in patients under middle age with sound hearts. We shall warn the patient, however, or his nearest friend,[2] that in angina only too frequently

[1] Broadbent, Sir W., *Lancet*, May 27, 1901.

[2] As for the patient himself perhaps κέοδος ἐν κακοῖς ἀγνωσία.

death is sudden, often at the beginning of an attack; but, if long deferred, the event is more often by way of ordinary heart failure. Gairdner, it is true, considered death to be an essential term in the accurate definition of angina pectoris, yet death is no more essential to the conception of angina pectoris than of the plague. Some recent writers on angina, in pardonable reaction against the counterfeits of the disease, dwell on its "almost invariable fatal termination." They declare that about angina pectoris there must be no mistake. It is indeed no disease to be trifled with, but we must not be compelled by modern necrologists into too gloomy a prognosis. I have argued in many paragraphs that in acute cases in young people, whose coronary circulation may be unimpaired, recovery, however grievous the seizures, is not the exception, but rather the rule; and even in old men, with degenerate arteries, recovery is not to be despaired of. The older writers, less enthralled by the dicta of the modern post-mortem room, less chastened perhaps by its discipline, recorded not a few recoveries, crude and tentative as their treatment necessarily was. As under chloroform: one patient, whose chances seem slender, escapes, while another, whose cardiac condition proves post-mortem to have been fair enough, succumbs. Desportes' *pronator teres* patient, although a bulky, gouty, sedentary clock-maker, suffering from typical attacks, completely recovered on the maintenance of a moxa in his thigh for 18 months. Heberden and Fothergill each relates a case of cure in patients æt. 30 and 35 respectively, *i.e.* in young adults. I have upon my notes the following extract from an author of the eighteenth century, whose name I have unfortunately mislaid, "experientia tamen morbum immedicabilem vocare non licet, quum plures angina pectoris laborantes perfectae sanitati restituti sint." A case of cure is also credited to Abercrombie,[1] in a girl æt. 13, but on reference to his report I cannot read the diagnosis as sufficiently definite; she had cardiac symptoms. Jurine collected no little evidence of recoveries, "even in old chronic cases," and uses them as an argument against the coronary hypothesis. Laennec speaks confidently of recoveries, even in severe cases.[2] A friend of my own, well stricken in years, and whose vessels

[1] Abercrombie, *Trans. Med. Chir. Soc.* [2] Laennec, *loc. cit.*, p. 350.

are probably as far advanced in senescence (he is not a patient of mine), suffered severely for two or three years from angina, both major and minor. At one period he could not walk fifty yards, however cautiously, without bringing on stenocardia. Happily under treatment the disease abated, and now for several years he has been restored to his eminent public functions, and is able without anxiety to address large meetings, and continually to answer no inconsiderable demands upon his strength of body and mind. Of late however he tells me that on severer efforts, which cannot always be avoided, some substernal tightness is prone to return. In other parts of this essay I have mentioned similar cases under my own care. Sir Lauder Brunton said, at the Belfast Meeting, that "cases (of angina)—even very severe ones—might apparently recover entirely, and remain well for years." This seems to upset the notion of cardiac degeneration; but such, by the way, is not his hypothesis.

With Dr. Lloyd Jones I saw Mr. P., æt. 53. Typical sternal and brachial pain radiating to lower jaw. Continually stopped by it while walking, "when he turns pale." No dyspnea. Grey complexion, and looks older than his years. Urine normal. Is sure he has had no syphilis, and there is no sign of it. Very moderate in alcohol and tobacco; and no great worries in home or business. Blood pressure, full systolic, 150. Slight systolic murmur at base, and clacking second sound. Accessible arteries a little thick. This patient—of whom I have often heard since from Dr. Jones—in time got well, so far at any rate as to consider his health good, to walk to business, and to do all his work.

A clear case of recovery under Dr. Bramwell has been alluded to already. Also in my own notes I find the following instance of recovery of a patient under the care of Dr. Atkinson of Saffron Walden:

Male, æt. 59, nervous, and of a neurotic family. Dr. Atkinson was called to him urgently on his first attack, which was typical and severe. There was neither then nor afterwards any sign of diseased heart or kidney; and so far as the finger could tell, no persisting rise of blood pressure. The arteries of the limbs were a little too palpable and visible. Sir William Broadbent, who also saw the patient, agreed with our diagnosis. He became unable to walk up any ascent, and had almost to give up the horse exercise to which he was accustomed. After a course of treatment lasting some time, in which Dr. Atkinson found 5-grain doses of iodide of potassium

to be effectual, he gradually recovered; and twelve years later Dr. Atkinson wrote in answer to my enquiry, "He is now very well if he would only think so."

This note touches a point of importance. In several other instances I have seen how the foreboding of angina, or the agony of it, had so unstrung the nerves of the patient that he did not dare trust to his regained freedom; tests of immunity are dreaded, and every uneasy or incidental sensation dwelt upon. Such is the shadow of the wing of the black hawk!

Some years ago, with Dr. Prince of Buntingford, I saw an old squire (*vide* p. 250) who from the age of 88 to 92 was subject to typical angina. He recovered under nitrites, of which drugs he became gradually independent. He died, æt. 95, of "bronchitis," but never had any sign or symptom of "heart failure." Again, Mr. Walker, formerly of Lowestoft, will remember a case of angina in a middle-aged man, who was suffering also from aortic insufficiency. (Syphilis denied, but there was a long alcoholic history.) The anginal attacks were very severe and very frequent. The alcoholic indulgence was stopped, and when I saw him again, a year later, the angina had ceased. Mr. Walker wrote to me much later (July 17, 1911) that the patient had continued to be quite free so far as the angina was concerned.

My colleague Dr. Humphry also will remember a case of typical angina (p. 163) which we saw together, a case in which the attacks were many, severe and unmistakable, but recovery was complete, and now for many years permanent.

The following case also offers a good instance:

Female, past the change of life, sent to me on June 7, 1906, by Dr. Forrest of Terrington. He was called to her eighteen months ago for angina. Soon afterwards the symptoms amounted to the "status anginosus," the paroxysms being almost continuous. Amyl gave little relief, and caused nausea. She was brought round under injections of morphine, followed by nitroglycerine, used very frequently, which, with strict precautions of life and diet, compassed a substantial amendment. But in February some social pressure had thrown her back, though not to the gravest state. Yet even now she is continually subject to attacks. The pain starts at midsternum, and radiates to the left shoulder and arm; the arm may become "numb"; sometimes the pain may drive into

both arms. The attack is attended with a sense of "fearful faintness." Attacks not rarely occur during sleep; she wakes with a "dream of fainting which is worse than the pain, though this is bad enough." Without my suggestion she spoke of the deathlike sensation. She was a woman of high spirit and courage, moved much in society, and had lived well, not denying herself a few glasses of wine with her meals. She had driven out a good deal, but taken little bodily exercise. The systolic pressure was 180. Dr. F. reported the pulse as usually about 60. Generally the pulse showed little disturbance, but in her worst phases the heart became irregular and almost showed signs of failure. This however was temporary. There was definite dulness at the manubrium, and a little to the right of it. To press the finger even lightly into the episternal notch causes instantly acute pain, something after the kind of the attacks. Pressure, freely tested, anywhere else discovers no hyperæsthesia. The arteries not tortuous, or notably thick. A systolic murmur at base. Second aortic loud and rather tympanitic. Urine always normal (Forrest).

I did not see this lady again till March 1914, when she called about another ailment. For some time after her last visit she had suffered a good deal from the angina, but with perseverance it was subdued. "Now for some considerable time has been wholly free from it." She did not seem to care to talk much about it, and evidently feared lest it should return sometime. However she was sure that she was then, and had been, wholly free, and was going about as she pleased. The murmur at the base still persisted; and the arteries showed some thickening. The manubrial dull area was unchanged. But now she could bear manipulation in the jugular fossa without the slightest discomfort. There seemed to be no history of any infection before the onset of the angina.

Dr. Mackenzie also speaks of "complete cessation of pain and permanent recovery." Mr. Brook of Lincoln narrated to me two cases of complete recovery from "true" angina pectoris, and one of these was in a gentleman of 70 years of age. This patient survived, in health, for ten years more, and then died suddenly of "heart failure." Samberger, on his large experience (*loc. cit.*), denies that recovery is a rare exception; a hopeless prognosis, he says, is justified only in cases with cardiac disease. After treatment many of his 73 patients recovered so far as to return to some work; a few were completely cured. Morgan (*loc. cit.*) reports a number of cases of complete recovery. Unfortunately too often in angina the heart is submitted, not, as

under chloroform, to one or two trials, but to a long and searching series of them; thus the chances of slipping from under the inhibition are reduced.

But too often these hopes, even confident hopes, are dashed by the recurrence of the disease after no inconsiderable periods of cessation. I have more than one record of this unlucky reiteration, in some cases after a patient, even for two or three years or more, had regarded himself as a free man again, and had returned to active life; yet even then there is room for hope. Dreyer [1] published a case in which for seven years after the chancre there were no symptoms. Then appeared a long course of them, especially ulcerative lesions of the skin and tongue and iritis. During this period angina pectoris set in, was arrested by specific treatment, and twice reappeared with the same good therapeutical effect. After the third series of attacks the Wa.R. became for the first time negative and the angina wholly disappeared. How long, however, syphilitic infection may hang about, for many a year and even into old age, is well illustrated by a case sent to me by Dr. Mathewson of Bromley.

Male, æt. 60. Thirty years ago rupia. Evidently an orderly and intelligent person. He placed himself systematically under specific treatment for five years. No reinfection. Eleven years ago (see p. 175) became subject to angina, first attack on cycling up a hill; although subject to it ever since, has kept it at bay. In typical attacks the pain used to be substernal, passing to left upper pectoral muscle and down inner arm; severe at left wrist; thorax compressed. Full attacks are now rare, but is subject to occasional angina minor. Aortic direct murmur, no regurgitation. Angor not mentioned.

In many cases a subacute phase of the aortic lesion lights up again as if to impress upon us the duty of every precaution against persistent hyperpiesis, hard work, and other causes of vascular strain; or old adhesions, under some tension, are tender. I read lately of a case in which, after a period of angina pectoris in early adult life, the patient, who in middle life had enjoyed good health and full power of work, was in late life attacked again by the disease, and fell a victim to it. In such cases one may postulate a succession of two independent

[1] Dreyer, *Med. Klinik*, May 4, 1913, quoted *Epit. B.M.J.*

attacks of aortic lesion, the earlier one at any rate probably infective; although the first attack may in some degree have prepared the ground for the second. It is unreasonable to argue, as it was argued in a recent medical journal, that in such cases the disease had persisted throughout, and had merely fallen into abeyance, so that a bad prognosis ought never to have been remitted. Such occasional coincidences pertain to a minority of cases, and may be independent. Furthermore, we should be careful as clinical observers to ascertain, in such a recurrence of cardioarterial disease, that the death was a death by angina. About ten years ago, in consultation with the late Mr. Hyde Hills of Cambridge, I saw a gentleman of middle age, who was attacked by angina pectoris of some severity. In the course of a year or two he got well and returned to his ordinary occupations. Four or five years later he fell ill of heart disease and died; but, as we learnt on careful enquiry, not with the symptoms of angina pectoris, but with those of ordinary cardiac failure, due in all probability to coronary occlusion and associated myocardial degeneration; no uncommon sequel. As Dr. Graham Steell has said (*loc. cit.*), the angina may subside while symptoms and signs of myocardial failure run their ordinary course to death. Our colleague Lucas Champonnière, whose death we are lamenting, had suffered, it appears, from angina pectoris "some years before," but "by strict precautions" had been long free from it. One who at a certain stage of cardiovascular disease has suffered from angina pectoris may at a later stage die suddenly of heart disease; but, unless there were definite clinical evidences of a return of the angina, it would always be precipitate, and often erroneous, to attribute the death, as so commonly in such cases it is attributed, to this malady. Angina, although frequently associated with the various kinds of heart failure to which old people are but too prone to succumb, creates no immunity against them; and in earlier life an infection may set up not angina only but also a cardiac lesion to end fatally; as in the undergraduate of Selwyn, referred to, p. 274, whom I saw with Dr. Christian Simpson. From his acute rheumatic angina, terrible as this was, he recovered completely; but ten years later he died suddenly of the aortic valvular lesion originally associated with it. It may be taken as a maxim that

cases originating in an infection offer a more hopeful prognosis. In these cases especially, prognosis depends upon early diagnosis and prompt and skilful treatment (see case, p. 529). Syphilitic angina often issues in relative recovery, in disappearance, that is, of the angina; though always with permanent injury to the vessel itself, and only too often with permanent destruction of the aortic valve. But how, while the local lesions progress, by corrosion of sensory areas or alteration of stresses, anginal attacks may cease is illustrated by a case of Curschmann's (*loc. cit.*), in which long continued attacks of angina wholly vanished ("vollständig wieder verschwanden"), but bradycardia set in. After death, at the autopsy, bulging of the ascending arch was found, with disease of the coronary arteries and fibrous degeneration of the myocardium, involving, no doubt, the auriculo-ventricular tracts.

I have said in the foregoing pages that I regard angina with more hope than do many of my colleagues; that agonizing, appalling, as it is, its mortality partakes of the nature of an accident, the accident of an inhibition of a heart often already frail or undermined; and that even a healthy heart may, rarely, succumb under the inhibition of an intensely irritated afferent nerve: many such cases are on record. I have mentioned one, of a lady, not more than 30 years of age, and otherwise healthy, who died under such inhibition in the agony of a gall-stone. A decaying heart, one which under comfortable circumstances might carry on life for years, is far more liable to be upset by an inhibition so intense as an attack of anginal pain may compass; so that, as Latham said of this malady, "A man may often with stricter propriety be said to be ill of his symptoms than to be ill of his disease; and, what is more, to die of his symptoms than to die of his disease." The vagus nerve does not lose its inhibitory powers *pari passu* with deterioration of the cardiac muscle; a degenerated heart perhaps makes a stronger appeal for protection at all hazards, even to be nursed into death; it seems to be inhibited, not less but more readily. In denser "heart block" the vagus effect cannot, it is true, pass so actively from the auricle to the ventricle; but in partial block its influence is still manifest enough to become fatal; it is felt until dissociation is complete.

Here recurs the opinion that there is no close relation between the peril of death and the severity of the seizure. A slight seizure may prove fatal without warning either to patient or physician; as Sir William Osler graphically writes, "a stony stare, a slight change in facial expression, two or three gasps, and all is over." In three of this author's cases death occurred in the first attack—attacks assailing, no doubt, hearts already degenerate.

Of the several symptoms, I am disposed to think the degree of "angor" to be the most ominous. Angina minor, if without angor, may hang about for many a year without cutting the thread of life; and may ultimately disappear.

In some cases the disease remains stationary, or variable, for many years. Osler (Lumleian Lectures) cites 10 cases extending each over ten years; Norman Moore (*loc. cit.*) quotes one of this duration, and alludes to others. Forchheimer relates that not a few of his angina patients survived for eight or ten years; one survived for seventeen years.[1] John Hunter's attacks lasted over twenty years, increasing in frequency, and latterly recurring almost daily. Mr. Edmund Owen has pointed out that Hunter's angina was probably due to the experimental inoculation of syphilis when he was about forty years of age. A well-known man of business in Cambridge was subject to typical angina for eighteen years; ultimately he died in an attack. A lady whom I used to see from time to time in Yorkshire must have suffered for nearly as long a period. In June 1885, with Dr. Hebblethwaite of Cheltenham, then of Harewood, I saw G. Y., stud-groom. He had typical and very severe angina pectoris. There were then no physical signs of cardio-arterial disease. On August 4, 1911, in answer to my enquiries his son kindly wrote to me to say his father died on October 10, 1905, aged 78; the death certificate being "Hemiplegia and senile decay." He had remained subject to the angina, "if hurried in any way," to the end of his life, though he did not die of it. Before our consultation the attacks had been a terrible fight, but the nitroglycerine then ordered (1885) had "never failed since to give instant, or almost instant, relief. He was never tired

[1] Forchheimer, *Illinois Med. Journ.*, May 1910 (epitome in *Arch. des mal. du cœur*, Feb. 1911).

of blessing the day he began to use it." So again Dr. Hinde of Harrogate has reminded me of a gentleman whom I saw with him at Stockton for typical angina pectoris, and who lived in spite of it for twenty years longer, and then, at the age of 90, died of a hemiplegia. The angina, though it never vanished entirely, had much abated. If these stationary cases are somewhat exceptional, their number is nevertheless considerable; and if in the individual we cannot reckon upon these degrees of tolerance, the knowledge of the possibility may mitigate the severity of the physician's sentence.

Finally, can we by physical examination obtain any signs wherewith to guide our prognosis? Few, if any; by the orthodiascope, or more simply by percussion, we may discover a dilatation of the ascending aorta, perhaps therewith a basic systolic murmur, and a clacking second sound; but these and such signs are too frequent in old people to give us any special message concerning angina pectoris. Degrees of blood pressure are inconstant. Signs of cardiac failure—cardiac dyspnea, pulmonary crepitations—would of course alarm us in respect of life, but not especially concerning the angina.

CHAPTER IX

TREATMENT

A PHYSICIAN can have no greater reward than the knowledge that he has been the means of bringing relief in a cruel disease, especially when this relief prevails over an agonizing symptom. As in the older writers we turn over the chapters on the treatment of angina, and note the multiplicity, and on the whole the futility, of their vehement, often overvehement, endeavours to assuage the torture which defied their remedies, it is then that we realise the debt which we owe to Lauder Brunton for the discovery, in 1867, of the nitrites in the treatment of angina. Not one of us but has had volumes of thanks for our small share in handing on to some of the most tormented of mankind this ineffable charm. If the nitrites seem to be soothing rather than curative, yet we shall admit that by their use we are enabled to compass something more than a truce; while calming the transports of the sufferer and the grief of his friends, we liberate the functions which make for recovery; thus we gain not time and courage only but some therapeutical efficacy also, and advance, often far, on the way to more substantial amendment.

With the subsidence of the causes, the peril to life may pass away. Although in our text-books the section on the treatment of angina pectoris generally begins with the maxim that we must hold up a failing heart, I have tried to show that this maxim needs radical qualification. As Dr. Hertz says, tension is the only cause of true visceral pain. Usually, indeed, on the same page we are inconsistently warned not to administer the heart tonics of the digitalis class, but rather "by nitrites and the like to abate the energy of the heart."

But what is needed first, even in the angina of young persons, and more and more as age increases, is to protect the heart against inhibitive shock; and this is best attained by the more or less continuous use of *atropine*. Even in heart block, unless extreme, atropine retains some influence; and in case of pneumogastric irritation, as for instance by an encroaching adenitis, by atropine a pulse-rate of 30-40 may in 15 minutes be nearly doubled (Dehio and others). Vaquez reports that in a case of bradycardia "due to vagus irritation from an appendicitis," atropine at once removed the inhibition. Gerhardt, in a case of syncopic bradycardia due to irritation of the left vagus involved in a cancer of the tongue, found these attacks cut short by atropine.[1] Danielopolu,[2] judging by his experiments on slow pulse in jaundice and the like, tells us that our doses are often too small really to suspend the vagus. When the "tone" of the vagus is intense the dose of atropine should be 2 mg. (adult).[3] In a case of jaundice, by injecting 2 mg. he accelerated the pulse from 48 to 100 in the course of three-quarters of an hour. Moreover, if Eppinger and Hess are right, atropine, although it is said on the whole to raise blood pressure somewhat, perhaps by check of the vagus, dilates the peripheral and perhaps the coronary vessels.[4] To every sufferer then from frequent attacks, I administer atropine systematically, increasing the dose gradually as the system may be acquiring immunity; and besides its specific protective, the drug has also some sedative virtue.[5] It is said that atropine is of less effect in old persons; I doubt this, because in certain heart-block cases its effects are apparent, and in experiments upon myself I find it active enough. Professor Waller, as an antidote to vagus arrest under chloroform, recommended bromide of ethyl. There is no harm in giving lactate of calcium regularly on the chance that calcium ions, as opposed to potassium, may check inhibition (but see p. 526). When the attacks are infrequent

[1] Gerhardt, *Deutsche Arch. f. klin. Med.* Bd. cvi., 1912.

[2] Danielopolu, *Soc. de Biol.*, 5 juin, 1908.

[3] See Hecht u. Nobel (of von Pirquet's laboratory, Vienna), *Zeitschr. f. exp. Med.* Bd. i. He. 1 u. 2, 1913.

[4] Eppinger u. Hess, *Zeitschr. f. exp. Path. u. Ther.* vol. v. p. 3.

[5] It is said that the methyl-bromid of atropine is a useful form of the drug; I have not yet tried it.

the application of the remedy is less convenient. Conversely, it is indiscreet during an attack to apply smelling-salts or other peripheral irritants whereby the heart might be more exposed to cumulative reflex inhibition.

If, as frequently happens, the angina is associated with *gout*, we must counteract this part of the case, whether by spa treatment or, at home, by appropriate regimen and medicines, including colchicum, or by both methods; much will depend on the conditions of particular cases.[1] If the arterial pressures are abnormally high, and in all they are higher than the morbid susceptibility of the suffering parts can tolerate, so in every case means must be taken to moderate pressures. Baths and technical exercises therefore are ordinarily out of the question; unless in a quiescent interval, when they should be instituted with the utmost caution. I can understand that, instituted with such precautions, the Swedish methods may, in certain phases of certain cases, have some efficacy.

I am better aware of the usefulness of the *high frequency current*; under this method I have seen some cases of the lesser stenocardia (a. minor), with high blood pressure, if not permanently cured, yet substantially relieved, even for years; for to this means high pressure without angina is amenable (See p. 110). A patient under the care of Dr. A. Roberts of Harrogate and myself for high pressure with angina minor, thus received over a period of six years continuous benefits. In this case the course, which included, it is true, mild sulphur baths and waters, needed some three or four repetitions; but the effects of the later courses were more enduring. Ultimately however angina reappeared in more typical form and defeated all remedies. For angina major I have not made a full trial of it. A good deal has been written at home and abroad concerning the virtues of the high frequency current in angina, but the evidence is very conflicting. Dr. Humphris, evidently from no little experience, has spoken favourably of the method, but chiefly as a moderator of high pressures.[2]

[1] See Ebstein, *Gicht*, p. 257.

[2] Humphris, F. H., Section of Balneology, etc., of Roy. Soc. Med., session in April 1913. See also *Arteriosclerosis* (p. 112).

Rest.—When the malady first appears decisively, and still more during periods when the attacks are frequent, the patient should be advised to keep his bed for a while, and injections of morphine and atropine may become imperative; but if the attacks are slight and the recurrences infrequent, it is difficult to urge counsels of perfection; moreover, such a confinement might entail countervailing disadvantages. To put a very old person to bed for some weeks may mean his never getting up again; and even the elderly are often depressed by it; the appetite and digestion fall off, crepitations appear in the lungs, the muscles deteriorate, and prostatic troubles increase. I am content to say: Never bring on the pain; every renewal of it keeps up the intolerance. If for this end absolute stillness in bed be required, then bed for a while it must be, with a corresponding reduction of food. Thus, if at first the attacks be not abolished, they will be mitigated, and usually taper off. The subsequent imprisonment must be determined by the sagacity of the physician, guided by the sensations of an intelligent patient. Pawinski (*loc. cit.*) insists likewise on rest; in mild cases, he says, the confinement need not be irksome; but in severer cases "absolute rest for weeks, even for a year, may be imperative"—imperative, that is, if the patient think it worth while. A man of middle age may submit to conditions which an old man may not think worth while. For two severe cases I prescribed recumbency for long periods in the fresh air, after the manner of the sanatorium, and with no little success. It is for this reason, if for no others, that Pawinski demurs, as I have done, to the use of baths; either forbidding them entirely, or using them rarely and cautiously at neutral temperatures (p. 103); advice which Fiessinger [1] has recently emphasised. Landouzy and Heitz however at Royat do not hesitate to use the CO_2 baths, even in angina if with high blood pressures, to reduce "peripheral pressure" (? resistance); they declare that at the end of the "cure" radioscopy, front and oblique, may exhibit a recovery of the diameter of the aorta by one-fifth or one-fourth. They find the risks negligible. In ten years, with 200 anginal patients they have had only one sudden death during the treatment. They are able to obtain a "vaso-

[1] Fiessinger, *Sem. méd.*, 30 nov., 1910.

dilatation très douce" which eases all off so that things go smoothly. Heitz advises these baths in the angina of chronic aortic disease and warns against them in coronary angina, a fanciful distinction. Tripier (*loc. cit.*) urges the anginous to avoid all effort, "et garder un repos absolu à la chambre—avec alimentation légère, et point de tabac." The return to life should be very gradual. Shorter rests, as of four or six weeks in bed, while, as generally admitted, they reduce blood pressures, and give time for the lesion to gain some repair, may be cloying to the body or disheartening to the mind; much depends on the patient's temperament, elderly persons bear such confinement better than the young. The practical rule is that whether in bed, shelter, or reclining-chair, the attacks are, if possible, to be prevented, for each attack rises again and again from effect to cause. The principle and practice of rest in angina are then much the same as in aneurysm, and in such junctures a serene habit of mind is a blessed aid. The patient will instinctively find out the position of greatest ease. In some, even severe, cases I have observed that the patient found a little relief in raising the arms above the head. Peter Joseph Frank taught [1] that throughout the course of angina the left arm especially should be kept at rest.

Diet.—A certain anginous patient exclaimed, "I should soon be all right if I could live without eating." The sympathy in angina between the attacks and the functions of the stomach have been discussed already (p. 343). We hear from enthusiasts that recoveries from angina pectoris have been compassed by a dietary so careful and ingenious as to "reduce coronary arterio-sclerosis"; such cases, illustrated by X-ray photographs, would be full of interest. However, by exclusion of purins, and by moderation of high feeding, much may be done to abate angina; especially in the minor form so often dependent on high pressures. Our forefathers relied greatly upon the same discreet and abstinent diet as we should now prescribe. They also insisted that the diet must be such as to keep down as much as possible the flatulence from which these patients suffer; that the carbohydrates, especially bulky farinaceous and green foods, should be reduced, and liquids at meals restricted. The meals

[1] Frank, P. J., *Traité de path. interne*, vol. iv. p. 460 (1857).

should be spare and frequent. Parry reduced both food and wine, gave gentle laxatives, wisely economised the bodily warmth, and preached equanimity. Fothergill was bolder; he "cured" a case by vegetarian diet and gentle horse exercise. In all cases full meals and late meals should be carefully avoided. In nocturnal angina, as in asthma, a meal after midday should be of the lightest and simplest; such as a little boiled fish with dry toast or biscuit. In obese persons some cautious means should be taken gradually to reduce the fat. For flatulent, so-called "gouty" dyspepsia, no remedy is better than the old-fashioned mixture of soda, rhubarb, ginger, cardamoms, and gentian. Dr. Verdon, in troublesome "gastric" cases of angina, recommends the "Salisbury diet." All physicians of experience warn us in angina against a loaded colon, and recommend mercurials occasionally, and more frequently podophyllin in frequent small doses (say $\frac{1}{10}$ gr. thrice daily). Sir James Barr thinks that in angina the heart has too much calcium ions, therefore he eliminates lime from the drinking water and food, and restores the phosphoric acid and acid fruits recommended by many physicians of the past, and adopted by Desportes. He records a case in which on this system much relief was obtained; that is, the rate came down from a dozen attacks a day to one in three or four days; but he is careful to add that the patient was also confined to bed. The older notion was that by these means the "ossification" of the coronary arteries might be counteracted; Barr, as Desportes before him, claims for the method a still larger efficacy.

If lime be injurious, the milk cure advocated by Karell and others may be inadmissible. The Karell cure (p. 91), which in angina I have never attempted, consists in a week's rest in bed on a diet of 1000 cc. of milk daily; it is recommended in cases of frequent mild attacks in persons of overloaded stomach and inflated abdomen. In a case complicated with a foul colon and "torpid liver," if resorted to for this short time only, and together with strict bodily rest, it may be efficacious; but in the many cases of puffy, big-bellied persons with flabby hearts, I should regard it as an unfit, or even a perilous, method.

In one or two cases on my notes, in one patient especially under the care of Dr. Ralph Wilson of Kew, the lactic acid

bacillus method of treatment, addressed, as we are informed, to toxins in the bowels, was at least coincident with a remarkable amelioration, perhaps a cure, of a case of severe "stenocardia" with high pressures. With the "propter hoc" I will not meddle. The precautions in respect of the digestion are to be sedulously imposed and noted, for indeed a windy stomach, if not a primary cause of angina, may suffice to part the slender threads of life. At least the patient must rest both mind and body for an hour before as well as after his meals; if convenience permit, in recumbency.

Exercise, then, must be restricted to gentle walking, and on the level only; for ascent of the gentlest incline may determine an attack. The bedroom must be on the ground floor, and it is better to take the air in a low, easy carriage. The rule as to exertion is that nothing should be done which might induce an attack. Every seizure means a peg down in our progress. Even mental occupation should be restricted to routine duties, mental excitement being jealously averted; and, as an old author writes, "Bacchi denique et Veneris consortium ceu pestem fugiant aegroti."

It is difficult to see one's way in angina pectoris to the use of systematic exercises, active or passive. At the outset of the effort the pressures must rise, if to fall later. When the attacks are not frequent, trials might with great caution be attempted in quiet intervals. I have no personal experience of any such trials, but were I to make the attempt, I should do it on the Zander method, in which no effort of the will or even of the attention is required. On account of the irritability of the vagus (p. 522), as Dr. Walford has said, the neckbands should be quite loose.

As to *drugs*, a course of the iodides is the accepted routine, and, even if the case be not syphilitic, the doses may be carried cautiously to the extremer quantities. Sir Lauder Brunton thinks no drug wards off angina pectoris so effectively as full doses of iodide of potassium, or other preparation of iodine; but to appreciate the virtue of these medicines and their doses, the syphilitic must be distinguished from the non-syphilitic cases. Dr. Janeway gives the iodide in small doses over a very long period. For my own part I prefer iodide of sodium in angina, lest a

potassium salt should forward inhibition, perhaps a whimsical fear. The salt should be largely diluted in water or, as at Bath, in mineral water. Syphilis must be carefully enquired for, and on any reasonable suspicion specific treatment vigorously instituted. Salvarsan is said by many observers, as by Barié (*loc. cit.*) and Weintraud,[1] to relieve the angina of syphilitic aortitis very quickly; but it must not, in my diffident opinion, be trusted alone, nor without much precaution. I am told that the intravenous injection is followed by a drop of arterial pressure (10-20 mm.) so sharp that it might in case of vagus lability or closed coronaries be perilous. Of course in these cases all "ambulant" treatment is to be deprecated. In this infection I have most experience of mercurial inunction, after the method of Aix-la-Chapelle, now practised at Harrogate and elsewhere; by this method I have seen syphilitic angina promptly cured, and, generally speaking, without relapse of the angina. Curschmann has published[2] an interesting case of syphilitic angina thus relieved:

A woman aged 32 (previously under his care for syphilis). While again under a relapse, she was suddenly seized with severe angina pectoris and dyspnea, and she had a painful node upon the manubrium and another on the right radius, 2 cm. above the wrist. Physical signs negative, but the author suspected gumma about the base of the heart (aortitis ?). She was submitted to 30 inunctions during 3 months, when she was quite freed both from symptoms and signs. The nodes also had vanished.

In a few recent cases I have advised patients of this class to submit to mercurial injections, and in one case of specific aortitis under continuous observation, with permanent relief to the angina. Moreover after the course was finished the Wa.R., repeated three times, was negative. Aortic insufficiency remained.

In the case under the care of Dr. Walker of Lowestoft (p. 268), specific treatment was very successful.

Male, æt. 48, suffering from severe and typical angina pectoris; the pain originated under the upper sternum, presented the usual radiations, and the attacks were very frequent. When I saw him

[1] Weintraud, *Ther. d. Gegenwart*, Okt. 1911.

[2] Curschmann, "Arbeiten aus med. Klinik, Leipzig," *Pathol. d. Kreislaufs*, 1893.

with Dr. Walker, he told me that under his observation a systolic murmur had appeared first, then a diastolic ; then the heart dilated, and pressure fell, on which account digitalis had been given for a short time, and with advantage. I also found a double aortic murmur ; the diastolic murmur—down the left border of the sternum—very loud. Under active specific treatment, Dr. Walker found the diastolic murmur first and then both murmurs to diminish ; then the diastolic wholly disappeared, and the systolic grew less and less. Meanwhile the attacks of angina, always amenable to the nitrites, abated, so that the patient could sit up in bed and begin to move gently about. The heart pulled itself together. In a few weeks the angina was at an end, though a weary aching about the chest continued for a long time afterwards (adhesions ?). I saw the patient again some time later, when he was fairly well ; the heart then was normal in all respects except a very soft systolic murmur and a dullish area to be detected at the manubrium and a little to the right of it. The patient denied syphilis, probably in good faith ; but there could be no doubt about the nature of the illness (cf. case, p. 179).

However, salvarsan, if the kidneys be sound, and the heart not gravely affected, may be better still ; Vaquez and Lantry,[1] in 28 cases of syphilitic aortitis, speak highly of its efficacy ; but whatever course be taken I would insist on promptitude. In syphilis the iodides are of course more than appropriate ; if the heavy doses are ill-borne, as only too often they are, Tripier gives the salt per rectum, dissolved in half a glass of water ; he gives two clysters of the solution, night and morning. Every two or three days he increases the doses by 10 or 12 gr. and continues this course for a month ; then he suspends it for a fortnight, repeating it thus as often as seems necessary. Some physicians find the new combinations of iodine (iodopin and others) better borne by their patients.

As syphilitic aortitis is no infrequent malady, and as perhaps the majority of instances of angina in patients of or under middle age are of this nature, Wassermann's test should be used in all such cases. In cases of this site and nature the percentage of positive results is very large (see case p. 210). Jacobaeus [2] used

[1] Vaquez and Lantry, *Soc. Méd. des Hôp.*, 7 juin, 1912.

[2] Jacobaeus, *Deutsche Arch. f. klin. Med.* Bd. cii., 1911. See also Stadler : protocol of discussion of a medical society, reported *Deutsche med. Wochenschr.*, 14 August, 1913.

this test in a large number of cases of chronic cardio-arterial degeneration; he excluded aneurysm and obvious gummatous aortitis, yet found notwithstanding that in a considerable proportion of the cases the response was affirmative. With improved technique the percentage is rapidly mounting up. A positive result indicates in suitable cases the immediate and active administration of antiluetic remedies.

Arsenic is credited, and in my opinion justly, with an alleviating effect in angina pectoris; it was used first, I believe, in this disease by Mr. Edward Alexander, a Halifax surgeon,[1] and at a later date was strongly recommended by Anstie. It is to be instituted in minute doses with meals, and gradually augmented even to some 15 or possibly 20 minims daily; but for short periods only, not more than a month or six weeks. I have not used doses so large. The bromides also are helpful, especially when the nervous centres have been exasperated by incessant pain. For continuous use the bromide of strontium is said to be preferable. Of their occasional use, to relieve attacks, I shall speak presently (p. 535). The nitrites are often administered, not only as palliatives to ward off or stop attacks, but continuously and systematically as a means of cure. It seems reasonable that by persistently abating aortic pressure, and so relieving stress, the lesion should have the better chance of repair. Sir William Osler, Ortner, and others with this view recommend doses of nitrite of sodium, or of Liquor Trinitrini, in increasing quantities as the case may need, even up to 20 grains or more of sodium nitrite per diem, or 15-20 m. of the solution; nay, in urgent distress, as we shall see, to larger doses still. For this increase the trinitrine is preferable to the sodium salt. I have no experience of these heroic doses; sometimes indeed sodium nitrite has appeared to me to be not without some toxic quality.

Generally speaking, digitalis and its kind are in mere angina to be eschewed; it is not easy to see any reason for the use of it, and it may raise arterial pressures and intensify vagus inhibition. We shall not agree then with the writer in Penzoldt and Sterzing's *System* (iii. 373), who proposes digitalis and caffeine, even during attacks; especially when we recollect that by mitral regurgitation angina pectoris

[1] Alexander, E., *Med. Comm.*, Dec. 3, 1789.

is assuaged. Yet, although this be the abstract view of the matter, it is true notwithstanding that in many cases, either on error of diagnosis, or deliberately for some special reason, digitalis and the cooler baths have been cautiously used without aggravating angina. In particular cases of failing heart the use of such measures as these must be left to the discretion of the physician, who will balance as best he can the several variables and their indications one against another.

Theobromine, in 7-10 gr. doses, is recommended by not a few writers (*e.g.* Marchiafava, Ortner, etc.) in the form of diuretin; whether for the thoracic or abdominal form it has many advocates, among whom I reckon myself. Janeway however has not much faith in it. It is said that when inserted into a vein in a moderate dose it lowers the blood pressure, apparently by splanchnic dilatation. Albu (*loc. cit.*) gives diuretin especially for the abdominal mode, 5-6 grm. daily for several days at a time; at the same time he administers the iodides freely. Dieulafoy recommends aspirin, in doses of 50 centigrammes every two hours; but only of course in urgency, for immediate aid.

When attacks are hanging about a carminative draught may expel the wind, to the great relief of the patient. In the lack of more specific remedies the physicians of a hundred years ago used these drugs abundantly: camphor, assafœtida, cajeput, musk, castoreum, valerian, oil of anise on sugar, and, best of all, in Fothergill's opinion, essence of peppermint. Once break the wind, they would say, and the attack is relieved. The following case, taken from a recent publication, was cited as "pseudo-angina pectoris" and attributed to flatulent distension of the stomach, "as the heart did not appear sufficiently diseased for there to be true angina!"

A middle-aged lady with a thick-walled, high-tension pulse had for some months been subject to "suffocative feelings" in the chest, coming on generally about an hour after meals. Then pain took the place of these feelings, and spread to the left arm. The attacks became more frequent, occurring five or six times a day and once or twice in the night, and had no relation to exercise. On examining the heart the apex beat was an inch outside the nipple line, but was forcible, and the sounds were good; the aortic second sound was accentuated.

Yet true angina no doubt it was. Dr. Verdon, whose interpretation of angina pectoris as a gastric affection I have discussed (p.406), says that to pass the soft œsophageal tube may, by discharging the load of the windy stomach, give prompt and precious aid; I think this operation should be attempted only in a patient inured to it, and protected by a dose of atropine. Flint reported of one of his cases that to swallow solid food would bring on the paroxysm. I have quoted Dr. Mackenzie's [1] advice to warn such patients against swallowing wind during efforts of eructation; and his observations on the influence of swallowing upon the vagus warn us to enjoin upon the patient to swallow as little as possible during an attack; he tells us that swallowing slows the sinus rhythm, and depresses the conductivity of the a-v fibres, so that their way may be blocked. The nerve supply of the œsophagus is mainly vagal (Langley). Herz of Vienna, advises the patient to take a small quantity of water into the mouth, then to throw the head back as far as possible in order to stretch the œsophagus, and at the same time to swallow, when the desired eructation will ensue. This attitude again, even if tolerable, may have its vagus perils. Mayer and Pribram showed that to irritate the wall of the stomach slowed the pulse and raised the blood pressure, effects which divisions of the vagus prevented. I find hydrocyanic acid a useful remedy in cases with much gastric disturbance, and Tr. Pruni Vergin. may conveniently be used for some weeks. Our audacious ancestors went so far as to give emetics in the attacks, an uncouth and forbidding, but possibly an efficacious remedy. Whether the sympathy between heart and stomach is only by mediation of the vagus, or is in part mechanical, we scarcely know.

It is unnecessary to dwell on the scrutiny of the various excretory functions, and the avoidance of gouty and other toxic elements of the diet, some of which may be vasoconstrictive.

More instantly, *to prevent the seizures*, the means at our service are first, of course, *the nitrites*. Atropine I have already mentioned (p. 522). The virtue of these agents is supposed to lie in their property of reducing the arterial pressures; this may

[1] Mackenzie on this point refers to Wyllie on Gastric Flatulence, *Edin. Hosp. Reports*, vol. iii.

be the explanation, as also of the antimonial draughts which our forefathers administered to the same end; nevertheless, it is not to be forgotten that the nitrites often relieve certain cardiac perturbations during which the pressures are moderate, perhaps by an active rather than a passive dilatation promoting the blood-stream. On the other hand by nitrite of amyl Dr. Mackenzie [1] promptly relieved an attack of angina in a man whose blood pressure was at the onset 190; though soon after the passage of the attack it proved to be 200. In a case, of which I have some notes, it momentarily reduced a pressure of 200, and the attack was checked, but the pressure soon rebounded to 230. These substances may possess some sedative or regulative virtue imperfectly known to us. In angina with aortic regurgitation, in which disease the periphery is usually fully expanded already,[2] the nitrites often give substantial relief nevertheless. It is often stated, in the confident way in which people propound unproved opinions, that the nitrites relieve angina by dilating the coronary arteries. That, unless in poisonous doses, they have any effect upon supposed constrictions of the coronary arteries is mere fancy. No one knows that they have this effect in the body; on the contrary, large peripheral dilatations elsewhere make it improbable. We have seen that the activity of these vessels depends directly on cardiac metabolism.

In the jaws of a seizure, nitrite of amyl is often inhaled from the crushed capsule, and the vapour, if not so certainly efficacious as nitroglycerine, answers, when it does answer, more quickly. In some persons it acts unpleasantly, and is not efficacious; it may be that in them irritation of the mucous membrane (fifth and tenth nerves) causes a reflex rise of blood pressure; if so, whatsoever reduction of pressure it may effect may be lost in a rebound; such persons might try the drug by the mouth; although, by the way, we have noted that in an attack the patient should swallow as little as possible, lest he intensify inhibition. However, because it is not wholly free from other irritating ethers (Stokvis, Albert Abrams, Vaquez),[3] amyl nitrite is falling out of favour. The

[1] Mackenzie, *Brit. Med. Journ.*, Oct. 20, 1906.
[2] See, however, Mackenzie's case, p. 335.
[3] *Arch. des mal. du cœur*, janvier 1903.

objections to chloroform, sometimes recommended in the status anginosus, I have considered (p. 483), it may imperil the heart; in cases of great urgency Sir Richard Powell prefers to inject m.j. of the standard nitroglycerine solution, with a little ether, subcutaneously.

Nitroglycerine by the mouth acts in about ten minutes; it is generally efficacious and well tolerated. The tablets are convenient for the pocket, but the alcoholic solution acts more quickly and surely. As much as 10 minims of the Liquor trinitrini (1 per cent solution) may have to be given to an accustomed patient at the outset of a severe attack. I have quoted Sir W. Osler's opinion that nitroglycerine is used much too timidly, and then dropped as ineffectual; but the unaccustomed patient is to be warned that the immediate effects of much smaller doses may be flushing and headache. These effects are harmless, do not mean pressure on the brain, and in time of need must be tolerated. To keep the periphery open, the nitroglycerine may be needed every two or three hours, when some increasing tolerance of the drug may have to be reckoned with. Erythrol tetranitrate, which for instant relief is less effectual, is considered more proper for continuous use, its pressure-reducing effect being more abiding; its scope is restricted by its higher cost, and it is said to be not without some toxic quality. However Dr. Churton of Leeds, in 1912, mentioned a case of angina pectoris of four years' standing in a man, aged 51, who had taken erythrol tetranitrate (gr. $\frac{1}{2}$) every six hours for fifteen months with great benefit. Unless the case be a severe one, and instant prevention imperative, reduction of pressures during some considerable part of the day may be sufficient to soothe the aorta, or at any rate to mitigate the sense of distress. With us all, pressures are fluctuating widely from hour to hour; and, as it is found by experiment that in the rabbit to raise aortic pressure considerably for short periods only will induce aortic lesions, so conversely it may be presumed that temporary reductions of pressures may soothe a damaged vessel. In mixed cases, in cases of angina with dyspnea and other cardiac symptoms, the nitrites are, on the whole, of less service, and if they reduce cardiac energy may even do harm. Alcohol as a vasodilator may be

effectual; thus it is said that a glass of hot grog at bed-time may avert nocturnal attacks.

If the attack be prolonged, a hot foot-bath, and hot fomentations or mustard poultice to the chest, are often comforting; Dieulafoy, on the contrary, has found an ice-bag, a means I should hardly venture to try, to give immediate ease. In past times the physician busied himself to evoke an attack of gout in a limb, and to this end used hot foot-baths, mustard, and the like; curious instances of relief by such methods are recorded in the older books. "Champagne and a tight boot" is, I am told, the modern form of the prescription.

A soporific named "Somnoform" is said to suspend the peripheral cardio-inhibitory fibres, and, if this be so, in severe and protracted seizures it may be both palliative and protective. Ethyl bromide is credited with the same discriminating virtue, and may be tried as a prophylactic. From chloral I shrink in these cases as I do from chloroform. In one case I saw the severe pain assuaged by rather liberal doses of antipyrine, but this drug again is one I should myself regard with some mistrust. I have said that in case of frequent worrying attacks, in which the nervous system is quivering under pain and insomnia, ammonium bromide in 20-grain doses may be prescribed with advantage. In one such case Dr. Mackenzie succeeded in blocking the pain by stupefying doses of bromide of ammonium.

Max Herz says that *oxygen inhalations*, administered for a short time two or three times a day, ward off stenocardia, improve the general condition, and even mitigate attacks. Some German physicians speak well of Finsen's light bath. Certain outrageous methods, such for instance as scalding the chest, front and back, with effusions of water boiled at the bedside, the application of large blisters, and so forth, testify, I hope, rather to the fierceness of the disease than to the barbarity of the medical warrior.

In severe and prolonged attacks, or in the "status anginosus," if the state of lung and kidney permit, hypodermic injection of *morphin*, or of morphin with atropin, is the most efficient means we have. It is said that in dragging (p. 483) operations morphia is a completer block than atropine. Heberden and

his successors used opium in some form or other, such as the aromatic tinctura thebaica, or laudanum. Latham used to give laudanum (10-30 m.); von Dusch relied on opium, castoreum and camphor; and many authors of experience have attributed to opium (or morphine) not only palliative, but also more permanent service. In modern practice of course morphine by subcutaneous injection (usually and rightly with atropine) is our way of using the opiate, and in an attack of any severity it is worse than useless to palter with the doses; a quarter of a grain should be given at once and, if required, a sixth more and more, at intervals of a quarter of an hour. Pain so exceeding carries off much morphine. Michaelis[1] truly says that "in many attacks we cannot get through without injection of morphine." He adds, "I have never seen death due to it, but the best results all round." When in very severe and pertinacious assaults the nitrites fail to be of service, no very infrequent difficulty, then morphine is our only, or our chief, resource. Morphine pulls the heart together, strengthens the beat and opens the peripheral circulation. It does also priceless service by the psychic peace which it induces. Ortner [2] prefers to morphine a form of opiate called pantopon; this preparation I find, in Merck's *Catalogues* for 1908–9, is an artificial combination of the alkaloids of opium. It is said to act more rapidly, efficaciously, and pleasantly than morphine; and rather improves than spoils the stomach and appetite. 1 cc. of a 2 per cent solution is injected under the skin; or by the mouth $\frac{1}{6}$-$\frac{2}{6}$ths gr. may be given, in pill or powder. Fränkel economises the morphine by intermediate subcutaneous injections of heroin hydrochloride, half a syringe of a 1 per cent solution = 0·005 gramme ($\frac{1}{200}$th of a grain). It is said to retain its virtue without increase of dose.

The many stories of cure by *issues* in this as in many other diseases are remarkable. Desportes' clockmaker, to whom I have alluded (p. 512), though a heavy, sedentary, gouty man, completely recovered on the insertion of a moxa in his thigh, which was kept open for eighteen months or more. I may mention also the case of Dr. MacBride of Dublin (A.D. 1776) and of Dr. Darwin, cited by Dr. Knott, in which complete cures were wrought by

[1] Michaelis, *Ther. d. Gegenwart*, 1909, p. 567.
[2] Ortner, *Jahreskurse*, Feb. 1911.

issues, containing one or two peas, on the inside of the thigh. Such testimony cannot be utterly ignored.

Venesection, by Heberden and Robert Hamilton, was put aside as useless. Whytt and Percival however did not discard it "for proper subjects"; still, even if by reduction of high pressure it had proved to be as serviceable a remedy as might be expected, it was generally agreed that, whatever the instant relief, to anæmiate a patient favoured the recurrence of attacks. Therefore I have never used the remedy in angina, tempted at times as I have been. Leeches, or a wet-cupping on the front of the chest, a favourite old remedy, may be worth trying in an obstinate case. But "cures" in angina are apt to be deceitful. Liberal venesection is said to be effectual in pulmonary œdema. Drugs, such as strychnine, which exalt the central irritabilty, will as a rule be avoided.

It would seem strange to leave a disease of or about the heart without more than my former allusion to *Digitalis*. But the part of digitalis in the treatment of angina pectoris can never be large, and is rather dark and perilous.[1] Digitalis stimulates the vagus centre, at any rate at first; any use of it must therefore be preceded by atropine Those who believe that angina pectoris implies a degenerate myocardium should think twice before using a drug which in such states has a double edge. If it should make for increase of arterial pressure, and for a widespread constriction of the arterioles, it might aggravate the evil; but these qualities of it are now again in doubt. In my view that angina pectoris is usually due to a sore aorta, why use a drug which has no special value in disease of this vessel, and which by vagus and sympathetic influences might multiply its perils? Notwithstanding, there are conditions under which, if used with caution and vigilance, digitalis may do service. Assuredly while the patient is beset by anginal attacks we cannot use it; it can be used only when the disease is mild or quiescent, when, if not for the angina itself, it may be needed for some concurrent cardiac disease, such as in old people is not infrequently associated with it, and constitutes its peril. We may then consider certain observations

[1] A good review of this question will be found by Sir Lauder Brunton, *Brit. Med. Journ.*, Nov. 15, 1910.

and experiments, from the time of Traube and Niemeyer to this day, proving that under certain conditions, even of high pressure, digitalis has seemed even to reduce arterial tension. Or again by spurring the heart to make an extra push in a critical phase excessive resistance may be overcome, and velocity promoted. For my part I cannot suppose such a mode of relief to be often desirable; so sharp a call might awaken the angina, and thrust the patient into an attack. Mr. Verdon has seen digitalis used in several cases of angina, and thought that by its use the attacks were aggravated.[1] I have tried it in a few cases of angina with faltering heart and also in syphilitic cases, wherein one might reckon more or less on a sound heart, but it seemed to do more harm than good. However, in the important article of Schmidt, in *Pflüger's Archives* (vol. cxxv.), we are told, in contradiction of Gottlieb and Magnus and current legend, that digitalis may dilate the splanchnic vascular area; and Pick, at the Wiesbaden Medical Congress in 1909, confirmed and even extended this assertion. By plethysmographic experiments upon the limbs, O. Müller found no change in this vascular bed under digitalis, but as the cardiac output was enlarged, he agreed with His that there must have been dilatation somewhere; though one does not see why increased velocity should not make at any rate some part of the relief, a relief which we often see from the drug in granular kidney. A good many experiments indicate that with a constant arterial diameter the blood race may be accelerated; on the other hand, if under high pressures the left ventricle is driving at the utmost capacity, and after a while strain effects become evident, how are we to get more out of the heart, unless perhaps for a transient spurt? Thus it would seem that the relief must be peripheral, and we ask ourselves again if the views of Hasebroek, and Grützner (Vol. I. p. 51), on a peristaltic activity in the muscular arteries, can be true? Some observers have assumed a livelier metabolism in the various organs as the means of acceleration, but we have no knowledge of any such influence on nutrition by digitalis.

Finally, in case of apparent death in or after an attack, the first duty is to inject intravenously (for an adult) 2 mg. of atropine. Meanwhile as I have often suggested, *artificial*

[1] Verdon, H. W., *Lancet*, July 13, 1912.

respiration should be practised assiduously. If the atropine and anæmia of the bulb should abate the mastery of the vagus, the once inhibited heart might be coaxed to beat again. Dr. Morison tried the method once, in vain; but his was an unpromising case, one in which indeed cerebral hæmorrhage may have been present.[1] In cases of far gone cardiac decay, of course little can be expected; yet Broadbent (*loc. cit.*) quotes an extreme case, in which on artificial respiration the pulse rallied, and the patient, partially restored, endeavoured to speak: the rally, however, was but transient. Sir Lauder Brunton in one of such cases kept the heart going for an hour, but the patient failed to get hold on life.[2] Dr. Curtin (in a letter to me on seeing this recommendation) wrote that independently he had tried artificial respiration in a case of great jeopardy. "About fifteen minutes after the seizure began the pulse became very unsteady, dropping about one beat in four. The result surprised me; my attempts restored the pulse, and normal rhythm was with some pains restored." Sir Richard Powell, on the same principle, recommends wafting of oxygen from a funnel over the face of the exanimate patient. This might be used with artificial respiration.

Upon the strict physiological life, open secretions, and psychic tranquillity, so needful in these cases, I need not dwell at greater length; nor upon those measures, general and special, which are appropriate to any cardiac disease with which the angina may be associated. For some reflections on the adaptation of these general rules to individual peculiarities I would refer the reader to Sir William Osler's treatise on *Angina Pectoris*.

In conclusion, I find it necessary to repeat that success in treating angina pectoris must be based on a correct analysis of each case. So long as the minds of young practitioners, and not of these only, are unsettled by the writings of eminent authors, who into this class have thrown pell-mell all sorts of neurotic or toxic, as well as some cardiac, disorders, cases which resemble angina only in that they are attended with motley distresses and various pains in the thorax, we cannot hope for consistent therapeutics. Moreover, the

[1] Morison, *Cardiac Pain*, p. 28.
[2] Brunton, Sir L., *Lancet*, Feb. 23, 1912.

physician, for his further discouragement, is assured that a differential diagnosis of such maladies is, or in certain cases may be, very difficult; this difficulty I have tried to appraise. Anginous patients seek many opinions, and, as I have given some attention to the subject, my experience of the disease has been fairly large; yet I do not hesitate to repeat, with a view to treatment, that if the observer will clear his mind of all the confusing disputes about "anginiform pains," "vasomotor anginas," "pseudo-anginas," "true anginas," and the rest of it, he will find no more difficulty of differential diagnosis than there must be in some few of the obscurer instances of any disease whatsoever. Such difficulty as may occasionally arise will be with those cases in which the symptoms are so incipient, or so masked, or so imperfectly observed, that for a while the data are insufficient; yet even of these a little patience and common sense, and the accurate and patient use of modern methods of diagnosis, will enable us to form no idle opinion; then the proper treatment will present itself.

P.S.—At the last moment a paper on Angina Pectoris by Vaquez has appeared in the *Arch. des mal. du cœur*, mars–avril 1915, in which the author goes a long way towards the pathology of the disease advocated in these pages. Some interesting facts in support are recorded. He would have done better still had he not clung to a fanciful distinction between the angina of effort and that of rest; between, that is, attacks produced by effort and those which come on without effort—as at night (see p. 254). Morgagni's case, with necropsy (p. 328), is almost enough to testify decisively to the contrary. Vaquez pleads still for a cardiac causation at any rate for the attacks of rest—or, as he names it, of "decubitus." The cases he has in view are of course those—not a few—complicated with heart disease. But the same patient is often subject to attacks coming on now in the one, and now in the other, of these modes of onset. To other points of Vaquez's argument I have in the previous pages given full consideration.

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